DISTRICT OF COLUMBIA PAIN-CAPABLE UNBORN CHILD PROTECTION ACT

HEARING

BEFORE THE

SUBCOMMITTEE ON THE CONSTITUTION

OF THE

COMMITTEE ON THE JUDICIARY
HOUSE OF REPRESENTATIVES

ONE HUNDRED TWELFTH CONGRESS

SECOND SESSION

ON

H.R. 3803

MAY 17, 2012

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APPENDIX

MATERIAL SUBMITTED FOR THE HEARING RECORD

DISTRICT OF COLUMBIA PAIN-CAPABLE UNBORN CHILD PROTECTION ACT

THURSDAY, MAY 17, 2012

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON THE CONSTITUTION,
COMMITTEE ON THE JUDICIARY,
Washington, DC.

The Subcommittee met, pursuant to call, at 4:06 p.m., in room 2141, Rayburn House Office Building, the Honorable Trent Franks (Chairman of the Subcommittee) presiding.

Present: Representatives Franks, Chabot, King, Nadler, Scott and Quigley.

Staff Present: (Majority) Paul Taylor, Subcommittee Chief Counsel; Jacki Pick, Counsel; Sarah Vance, Clerk; (Minority) David Lachmann, Subcommittee Staff Director; and Veronica Eligan, Professional Staff Member.

Mr. FRANKS. This hearing will come to order. Thank you all for being here today. We especially appreciate our witnesses here. And without objection, the Chair is authorized to declare a recess of the Committee at any time. And again, we welcome you all here.

And I recognize myself now for 5 minutes for an opening statement.

The gruesome late-term abortions of unborn children who can feel pain is, in my opinion, the greatest human rights atrocity in the United States today. Today's hearing examines H.R. 3803, the District of Columbia Pain-Capable Unborn Child Protection Act. This bipartisan measure has greater than 190 sponsors in the House of Representatives, and it protects unborn children who can feel pain from being subjected to inhumane, torturous late-term abortions.

Medical science regarding the development of unborn babies and their capacities at various stages of growth has advanced very dramatically, demonstrating clearly that unborn children indeed experience pain. The biggest single hurdle to legislation like H.R. 3803 is that opponents deny unborn babies feel pain at all, as if somehow the ability to feel pain magically develops instantaneously as the child passes through the birth canal.

This level of understanding might be excused in earlier eras of human history, but the evidence available to us today is extensive and irrefutable. Unborn children have the capacity to experience pain at least by 20 weeks, and very likely substantially earlier.

I will now enter into the record a 29-page summary of the dozens of studies worldwide confirming that unborn children feel pain by

at least 20 weeks postfertilization. This information is available at www.doctorsonfetalpain.org. That is www.doctorsonfetalpain.org. And I recommend that all committee members, their staff, and members of the press review this site to get the most current evidence on unborn pain, rather than to have their understanding cemented in an earlier time when scientists still believed in spontaneous generation and that the Earth was flat.

[The information referred to follows:]

Fetal Pain: The Evidence

The eleven points below summarize the substantial medical and scientific evidence that unborn children can feel pain by 20 weeks after fertilization.

www.doctorsonfetalpain.org

posted March 14, 2011

1: Pain receptors (nociceptors) are present throughout the unborn child's entire body by no later than 20 weeks after fertilization and nerves link these receptors to the brain's thalamus and subcortical plate by no later than 20 weeks.

DOCUMENTATION:

a. Pain receptors (nociceptors) are present throughout the unborn child's entire body by no later than 20 weeks.

1. **Myers, 2004**, p.241, para.2, "The first essential requirement for nociception is the presence of sensory receptors, which first develop in the perioral area at approximately 7 weeks gestation and are diffusely located throughout the body by 14 weeks.[95]"

   Myers LB, Bulich LA, Hess, P, Miller, NM. Fetal endoscopic surgery: indications and anaesthetic management. *Best Practice & Research Clinical Anaesthesiology.* 18:2 (2004) 231-258.

   [95]Smith S. Commission of Inquiry into Fetal Sentience. London: CARE, 1996.

2. **Derbyshire, 2010**, p.7, para.2, "For the foetus, an existence of 'pain' rests upon the existence of a stimulus that poses a threat to tissue, being detected by a nervous system capable of preferentially responding to stimuli that pose a threat to tissue. The entire experience is completely bounded by the limits of the sensory system and the relationship between that system and the stimulus. If pain is conceived of in this manner then it becomes possible to talk of foetal pain anytime between 10 and 17 weeks GA [gestational age] when nociceptors develop and mature, and there is evidence of behavioural responses to touch."

   Note: Derbyshire's other published works indicate that he believes pain requires subjective human experience, not possible until after birth; nonetheless, he acknowledges this finding.

   Derbyshire SW, Foetal pain? *Best Practice & Research Clinical Obstetrics and Gynaecology* 24:5 (2010) 647-655.

3. **Anand, 1987**, p.2, para.2, "Cutaneous sensory receptors appear in the perioral area of the human fetus in the 7th week of gestation; they spread to the rest of the face, the palms of the hands, and the soles of the feet by the 11th week, to the trunk and proximal parts of the arms and legs by the 15th week, and to all cutaneous and mucous surfaces by the 20th week.[25,26]"

> Anand KJS, Hickey PR. Pain and its effects in the human neonate and fetus. *New England Journal of Medicine*. 317:21 (1987) 1321-1329.
>
> [25]Humphrey T. Some correlations between the appearance of human fetal reflexes and the development of the nervous system. *Progress in Brain Research*. 4 (1964) 93-135.
>
> [26]Valnaan HB, Pearson JP. What the fetus feels. *British Medical Journal*. 280 (1980) 233-234.

4. **Vanhatalo, 2000**, p.146, col.2, para.2, "First nociceptors appear around the mouth as early as the seventh gestational week; by the 20th week these are present all over the body."

> Vanhalto S, van Nieuwenhuizen O. Fetal Pain? *Brain & Development*. 22 (2000) 145-150.

5. **Brusseau, 2008**, p.14, para.3, "The first essential requirement for nociception is the presence of sensory receptors, which develop first in the perioral area at around 7 weeks gestation. From here, they develop in the rest of the face and in the palmar surfaces of the hands and soles of the feet from 11 weeks. By 20 weeks, they are present throughout all of the skin and mucosal surfaces.[19]

> Brusseau R. Developmental Perpectives: is the Fetus Conscious? *International Anesthesiology Clinics*. 46:3 (2008) 11-23.
>
> [19]Simons SH, Tibbool D. Pain perception development and maturation. *Seminars on Fetal and Neonatal Medicine*. 11 (2006) 227-231.

b. nerves link these receptors to the brain's thalamus and subcortical plate by no later than 20 weeks.

1. **Van Scheltema 2008**, p.313, para.1 — "The connection between the spinal cord and the thalamus (an obligatory station through which nearly all sensory information must pass before reaching the cortex) starts to develop from 14 weeks onwards and is finished at 20 weeks."

> Van Scheltema PNA, Bakker S, Vandenbussche FPHA, Oepkes, D. Fetal Pain. *Fetal and Maternal Medicine Review*. 19:4 (2008) 311-324.

2. **Glover, 1999**, p.882, col.1, para.1, "Most incoming pathways, including nociceptive ones, are routed through the thalamus and, as stated above, penetrates the subplate zone from about 17 weeks… These monoamine fibres start to invade the subplate zone at 13 weeks and reach the cortex at about 16 weeks. This puts an early limit on when it is likely that the fetus might be aware of anything that is going on in its body or elsewhere."

   Glover V. Fetal pain: implications for research and practice. *British Journal of Obstetrics and Gynaecology.* 106 (1999) 881-886.

3. **Lee, 2005**, p.950, col.1, "In contrast to direct thalamocortical fibers, which are not visible until almost the third trimester, thalamic afferents begin to reach the somatosensory subplate at 18 weeks' developmental age (20 weeks' gestational age)[16] and the visual subplate at 20 to 22 weeks' gestational age. These afferents appear morphologically mature enough to synapse with subplate neurons.[17]"

   Note: Lee et al. believe that pain requires conscious cortical processing, which they deem unlikely until 29 or 30 weeks; nonetheless, they acknowledges this finding.

   Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

   [16]Kostovic I, Rakic P. Developmental history of the transient subplate zone in the visual and somatosensory cortex of the macaque monkey and human brain. *Journal of Comparative Neurology.* 297 (1990) 441-470.

   [17]Hevner RF. Development of connections in the human visual system during fetal mid-gestation: a DiI-tracing study. *Journal of Experiemental Neuropathology & Experimental Neurology.* 59 (2000) 385-392.

4. **Gupta, 2008**, p.74, col.2, para.1, " Peripheral nerve receptors develop between 7 and 20 weeks gestation… Spinothalamic fibres (responsible for transmission of pain) develop between 16 and 20 weeks gestation, and thalamocortical fibres between 17 and 24 weeks gestation."

   Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Continuing Education in Anaesthesia, Critical Care & Pain.* 8:2 (2008) 71-75.

2: By 8 weeks after fertilization, the unborn child reacts to touch. After 20 weeks, the unborn child reacts to stimuli that would be recognized as painful if applied to an adult human, for example by recoiling.

DOCUMENTATION:

a. By 8 weeks after fertilization, the unborn child reacts to touch.

1. **Gupta, 2008**, p.74, col.2, para.2, "Movement of the fetus in response to external stimuli occurs as early as 8 weeks gestation…"

   Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Continuing Education in Anaesthesia, Critical Care & Pain.* 8:2 (2008) 71-75.

2. **Glover, 2004**, p.36, para.4, "The fetus starts to make movements in response to being touched from eight weeks, and more complex movements build up, as detected by real time ultrasound, over the next few weeks."

   Glover V. The fetus may feel pain from 20 weeks; The Fetal Pain Controversy. *Conscience.* 25:3 (2004) 35-37.

3. **Myers 2004**, p.241, para.6, "A motor response can first be seen as a whole body movement away from a stimulus and observed on ultrasound from as early as 7.5 weeks' gestational age. The perioral area is the first part of the body to respond to touch at approximately 8 weeks, but by 14 weeks most of the body is responsive to touch."

   Myers LB, Bulich LA, Hess, P, Miller, NM. Fetal endoscopic surgery: indications and anaesthetic management. *Best Practice & Research Clinical Anaesthesiology.* 18:2 (2004) 231-258.

4. **Derbyshire, 2008**, p.119, col.2, para.4, "Responses to touch begin at 7–8 weeks gestation when touching the peri-oral region results in a contralateral bending of the head. The palms of the hands become sensitive to stroking at 10-11 weeks gestation and the rest of the body becomes sensitive around 13-14 weeks gestation.[35]"

   Note: Derbyshire's other published works indicate that he believes pain requires subjective human experience, not possible until after birth; nonetheless, he acknowledges this finding.

   Derbyshire SW. Fetal Pain: Do We Know Enough to Do the Right Thing? *Reproductive Health Matters.* 16: 31Supp. (2008) 117-126.

   [35]Fitzgerald M. Neurobiology of fetal and neonatalpain. In:Wall P, Melzack R, editors. Textbook of Pain. Oxford Churchill Livingstone, 1994. p.153–63.

b. After 20 weeks, the unborn child reacts to stimuli that would be recognized as painful if applied to an adult human, for example by recoiling.

1. **Gupta, 2008**, p. p.74, col.2, para.2, "Behavioural responses… Response to painful stimuli occurs from 22 weeks gestation [= 20 weeks post-fertilization]."

   Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Continuing Education in Anaesthesia, Critical Care & Pain.* 8:2 (2008) 71-75.

2. **Giannakoulopoulos, 1994**, p.77, col.2, para.3, "We have observed that the fetus reacts to intrahepatic vein needling with vigorous body and breathing movements, which are not present during placental cord insertion needling."

   Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling. *Lancet.* 344 (1994) 77-81.

3. **Lowery, 2007**, p.276, col.2, para1, "Fetuses undergoing intrauterine invasive procedures, definitely illustrative of pain signaling, were reported to show coordinated responses signaling the avoidance of tissue injury.[15]"

   Lowery CL, Hardman MP, Manning N, Clancy B, Hall RW, Anand KJS. Neurodevelopmental Changes of Fetal Pain. *Seminars in Pernatology.* 31 (2007) 275-282.

   [15]Williams C. Framing the fetus in medical work: rituals and practices. *Social Science & Medicine.* 60 (2005) 2085-2095.

4. **Mellor, 2005**, p.457, col.1, para.2, "For instance, the human fetus responds to intrahepatic needling (versus umbilical cord sampling) by moving away and with an increase in the levels of circulating stress hormones. . .[71,72,74,75]"

   Note: Mellor et al. believe that the unborn child is kept 'asleep' in utero, *and therefore does not perceive pain; nonetheless, they recognize this finding.*

   Mellor DJ, Diesch TJ, Gunn AJ, Bennet L. The importance of 'awareness' for understanding fetal pain. *Brain Research Reviews.* 49 (2005) 455-471.

   [71] Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling. *Lancet.* 344 (1994) 77-81.

   [72]Giannakoulopoulos X, Teixeira J, Fisk N. Human fetal and maternal noradrenaline responses to invasive procedures. *Pediatric Research.* 45 (1999) 494-499.

   [74]Gitau R, Fisk NM, Teixeira JM, Cameron A, Glover V. Fetal hypothalamic–pituitary–adrenal stress responses to invasive procedures are independent of maternal responses. *Journal of Clinical Endocrinology and Metabolism.* 86 (2001) 104-109.

   [75]Gitau R, Fisk NM, Glover V. Human fetal and maternal corticotrophin releasing hormone responses to acute stress. *Archives of Disease in Childhood - Fetal Neonatal Edition.* 89 (2004) F29-F32.

3: In the unborn child, application of such painful stimuli is associated with significant increases in stress hormones known as the stress response.

DOCUMENTATION:

1. **Tran, 2010**, p.44, col.1, para.7, "Invasive fetal procedures clearly elicit a stress response…"

   Tran, KM. Anesthesia for fetal surgery. *Seminars in Fetal & Neonatal Medicine.* 15 (2010) 40-45.

2. **Myers, 2004**, p.242, para.2, "Human fetal endocrine responses to stress have been demonstrated from as early as 18 weeks' gestation. Giannakoulopoulos et al[99] first demonstrated increases in fetal plasma concentrations of cortisol and β-endorphin in response to prolonged needling of the intrahepatic vein (IHV) for intrauterine transfusion. The magnitude of these stress responses directly correlated with the duration of the procedure. Fetuses having the same procedure of transfusion, but via the non-innervated placental cord insertion, failed to show these hormonal responses. Gitau et al[100] observed a rise in β-endorphin during intrahepatic transfusion from 18 weeks' gestation, which was seen throughout pregnancy independent both of gestation and the maternal response. The fetal cortisol response, again independent of the mother's, was observed from 20 weeks' gestation.[100] Fetal intravenous administration of the opioid receptor agonist, fentanyl, ablated the β-endorphin response and partially ablated the cortisol response to the stress of IHV needling, suggesting an analgesic effect.[101] A similar, but faster, response is seen in fetal production of noradrenalin to IHV needling. This too is observed in fetuses as early as 18 weeks, is independent to the maternal response and increases to some extent with gestational age.[102] Thus, from these studies one can conclude that the human fetal hypothalamic– pituitary–adrenal axis is functionally mature enough to produce a β-endorphin response by 18 weeks and to produce cortisol and noradrenalin responses from 20 weeks' gestation."

   Myers LB, Bulich LA, Hess, P, Miller, NM. Fetal endoscopic surgery: indications and anaesthetic management. *Best Practice & Research Clinical Anaesthesiology.* 18:2 (2004) 231-258.

   [99] Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling. *Lancet.* 344 (1994) 77-81.

   [100] Gitau R, Fisk NM, Teixeira JM, Cameron A, Glover V. Fetal hypothalamic–pituitary–adrenal stress responses to invasive procedures are independent of maternal responses. *Journal of Clinical Endocrinology and Metabolism.* 86 (2001) 104-109.

   [101] Fisk NM, Gitau R, Teixeira MD, Giannakoulopoulos, X, Cameron, AD, Glover VA. Effect of Direct Fetal Opioid Analgesia on Fetal Hormonal and Hemodynamic Stress Response to Intrauterine Needling. *Anesthesiology.* 95 (2001) 828-835.

   [102] Giannakoulopoulos X, Teixeira J, Fisk N, Glover V. Human fetal and maternal noradrenaline responses to invasive procedures. *Pediatric Research.* 45(1999) 494-499.

3. **Derbyshire, June 2008**, p.4, col.1, para.5, "Another stage of advancing neural development takes place at 18 weeks, when it has been demonstrated that the fetus will launch a hormonal stress response to direct noxious stimulation."

   Note: Derbyshire believes that pain requires subjective human experience, not possible until after birth; nonetheless, he acknowledges this finding.

   Derbyshire SW. Fetal Pain: Do We Know Enough to Do the Right Thing? *Reproductive Health Matters*. 16: 31Supp. (2008) 117-126.

4. **Gupta, 2008**, p.74, col.2, para.3, "Fetal stress in response to painful stimuli is shown by increased cortisol and β-endorphin concentrations, and vigorous movements and breathing efforts.[7,9] There is no correlation between maternal and fetal norepinephrine levels, suggesting a lack of placental transfer of norepinephrine. This independent stress response in the fetus occurs from 18 weeks gestation.[10]"

   Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Continuing Education in Anaesthesia. Critical Care & Pain.* 8:2 (2008) 71-75.

   [7]Boris P, Cox PBW, Gogarten W, Strumper D, Marcus MAE. Fetal surgery, anaesthesiological considerations. *Current Opinion in Anaesthesiology.* 17 (2004) 235-240.

   [9]Giannakoulopoulos X, Teixeira J, Fisk N. Human fetal and maternal noradrenaline responses to invasive procedures. *Pediatric Research.* 45 (1999) 494-499.

   [10]Marcus M, Gogarten W, Louwen F. Remifentanil for fetal intrauterine microendoscopic procedures. *Anesthesia & Analgesia*. 88 (1999) S257.

5. **Fisk, 2001**, p.828, col.2, para.3, "Our group has shown that the human fetus from 18-20 weeks elaborates pituitary-adrenal, sympatho-adrenal, and circulatory stress responses to physical insults." p.834, col.2, para.2, "This study confirms that invasive procedures produce stress responses…."

   Fisk NM, Gitau R, Teixeira MD, Giannakoulopoulos, X, Cameron, AD, Glover VA. Effect of Direct Fetal Opioid Analgesia on Fetal Hormonal and Hemodynamic Stress Response to Intrauterine Needling. *Anesthesiology*. 95 (2001) 828-835.

4: Subjection to such painful stimuli is associated with long-term harmful neurodevelopmental effects, such as altered pain sensitivity and, possibly, emotional, behavioral, and learning disabilities later in life.

DOCUMENTATION:

1. **Van de Velde, 2006**, p.234, col.1, para.3, "It is becoming increasingly clear that experiences of pain will be 'remembered' by the developing nervous system, perhaps for the entire life of the individual.[22,33] These findings should focus the attention of clinicians on the long-term impact of early painful experiences, and highlight the urgent need for developing therapeutic strategies for the management of neonatal and fetal pain."

   Van de Velde M, Jani J, De Buck F, Deprest J. Fetal pain perception and pain management. *Seminars in Fetal & Neonatal Medicine.* 11 (2006) 232-236.

   [22] Vanhalto S, van Nieuwenhuizen O. Fetal Pain? *Brain & Development.* 22 (2000) 145-150.

   [33]Anand KJS. Pain, plasticity, and premature birth: a prescription for permanent suffering? *Nature Medicine.* 6 (2000) 971-973.

2. **Vanhatalo, 2000**, p.148, col.2, para.4, "All these data suggest that a repetitive, or sometimes even strong acute pain experience is associated with long-term changes in a large number of pain-related physiological functions, and pain or its concomitant stress increase the incidence of later complications in neurological and/or psychological development."

   Note: Vanhalto & Niewenhuizen believe that pain requires cortical processing; nevertheless, they acknowledge that, "noxious stimuli may have adverse effects on the developing individual regardless of the quality or the level of processing in the brain...after the development of the spinal cord afferents around the gestational week 10, there may be no age limit at which one can be sure noxae are harmless." (p.149, col.1, para.2).

   Vanhalto S, van Nieuwenhuizen O. Fetal Pain? *Brain & Development.* 22 (2000) 145-150.

3. **Gupta, 2008**, p.74, col.2, para.3, " There may be long-term implications of not providing adequate fetal analgesia such as hyperalgesia, and possibly increased morbidity and mortality."

   Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Continuing Education in Anaesthesia, Critical Care & Pain.* 8:2 (2008) 71-75.

4. **Lee, 2005,** p.951, col.1, para.3, "When long-term fetal well-being is a central consideration, evidence of fetal pain is unnecessary to justify fetal anaesthesia and analgesia because they serve other purposes unrelated to pain reduction, including ... (3) preventing hormonal stress responses associated with poor surgical outcomes in

neonates[71,72]; and (4) preventing possible adverse effects on long-term neurodevelopment and behavioral responses to pain.[73-75,,]

Note: Lee et al. believe that pain requires conscious cortical processing, which they deem unlikely until 29 or 30 weeks; nonetheless, they acknowledges this finding.

Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

[71]Anand KJ, Hickey PR.Halothane-morphine compared with high-dose sufentanil for anesthesia and postoperative analgesia in neonatal cardiac surgery. New England Journal of Medicine. 326 (1992) 1-9.

[72]Anand KJ, Sippell WG, Aynsley-Green A. Randomised trial of fentanyl anaesthesia in preterm babies undergoing surgery: effects on the stress response. *Lancet.* 329 (1987) 62-66.

[73]Johnston CC, Stevens BJ. Experience in a neonatal intensive care unit affects pain response. *Pediatrics.* 98 (1996) 925-930.

[74]Taddio A, Katz J, Ilersich AL, Koren G. Effect of neonatal circumcision on pain response during subsequent routine vaccination. *Lancet.* 349 (1997) 599-603.

[75]Taylor A, Fisk NM, Glover V. Mode of delivery and subsequent stress response. *Lancet.* 355 (2000) 120.

5: For the purposes of surgery on unborn children, fetal anesthesia is routinely administered and is associated with a decrease in stress hormones compared to their level when painful stimuli are applied without such anesthesia.

DOCUMENTATION:

a. For the purposes of surgery on unborn children, fetal anesthesia is routinely administered.

1. **Van de Velde, 2005**, p.256, col.2, para.2, "Therefore, it has been suggested that pain relief has to be provided during *in utero* interventions on the fetus from mid-gestation (20 weeks) on.[32-34]"

   Van de Velde M, Van Schoubroeck DV, Lewi LE, Marcus MAE, Jani JC, Missant C, Teunkens A, Deprest J. Remifentanil for Fetal Immobilization and Maternal Sedation During Fetoscopic Surgery: A Randomized, Double-Blind Comparison with Diazepam. *Anesthesia & Analgesia*. 101 (2005) 251-258.

   [32]Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling *Lancet*. 344 (1994) 77-81.

   [33]Giannakoulopoulos X, Teixeira J, Fisk N. Human fetal and maternal noradrenaline responses to invasive procedures. *Pediatric Research*. 45 (1999) 494-499.

   [34]Anand KJS, Maze M. Fetuses, fentanyl, and the stress response. *Anesthesiology*. 95 (2001) 823-825.

2. **Myers, 2004**, p.236, para.3, "The anaesthesiologist is required to provide both maternal and fetal anaesthesia and analgesia while ensuring both maternal and fetal haemodynamic stability…Since substantial evidence exists demonstrating the ability of the second trimester fetus to mount a neuroendrocrine response to noxious stimuli…fetal pain management must be considered in every case."

   p.240, col.5, "A substantial amount of both animal and human research demonstrated that the fetus is able to mount a substantial neuroendocrine response to noxious stimuli as early as the second trimester of pregnancy. Fetal neuroanatomical development further substantiates this research. Evidence also exists that suggests that these responses to noxious stimuli may, in fact, alter the response to subsequent noxious stimuli long after the initial insult. This is the rationale behind providing fetal anaesthesia and analgesia whenever surgical intervention is thought to potentially provide a noxious insult to the fetus."

   Myers LB, Bulich LA, Hess, P, Miller, NM. Fetal endoscopic surgery: indications and anaesthetic management. *Best Practice & Research Clinical Anaesthesiology*. 18:2 (2004) 231-258.

3. **Gupta, 2008**, p.74, col.2, para.4, "As with any procedure, the provision of analgesia depends on the likely severity of pain associated with the intervention. However, analgesia is recommended for:
   (i) endoscopic, intrauterine surgery on placenta, cord, and membranes;
   (ii) late termination of pregnancy;
   (iii) direct surgical trauma to the fetus."

   Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Continuing Education in Anaesthesia, Critical Care & Pain.* 8:2 (2008) 71-75.

4. **Giannakoulopoulos, 1994**, p.80, col.2, para.4, "Just as physicians now provide neonates with adequate analgesia, our findings suggest that those dealing with the fetus should consider making similar modifications to their practice. This applies not just to diagnostic and therapeutic procedures on the fetus, but possibly also to termination of pregnancy, especially by surgical techniques involving dismemberment."

   Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling. *Lancet.* 344 (1994) 77-81.

5. **Van Scheltema, 2008**, p.320, para.3, "Neuroanatomical, neurophysiological, hormonal, haemodynamic and behavioural data indicate that a fetus is capable of reacting to noxious stimuli, implying that the fetus can experience stress and possibly even pain… The changes described can be long-lasting, perhaps even life-long…We therefore think that when performing invasive intrauterine procedures it is important to accomplish fetal anaesthesia to protect the fetus from possible harmful effects on the developing neural system. It is difficult to determine from what gestation onwards fetal anaesthesia should be provided; however, we feel that it should be considered from at least mid-gestation."

   Van Scheltema PNA, Bakker S, Vandenbussche FPHA, Oepkes, D. Fetal Pain. *Fetal and Maternal Medicine Review*. 19:4 (2008) 311-324.

b. Fetal anesthesia … is associated with a decrease in stress hormones compared to their level when painful stimuli is applied without such anesthesia.

1. **Fisk, 2001**, p.834, col.2, para.3, "This study provides the first evidence that direct fetal analgesia reduces stress responses to intervention *in utero*."

   Abstract, "The authors investigated whether fentanyl ablates the fetal stress response to needling using the model of delayed interval sampling during intrahepatic vein blood sampling and transfusion in alloimmunized fetuses undergoing intravascular transfusion between 20 and 35 weeks.

   "Fentanyl reduced the β endorphin (mean difference in changes, -70.3 pg/ml; 95% confidence interval, -121 to -19.2;$P = 0.02$) and middle cerebral artery pulsatility index response (mean difference, 0.65; 95% confidence interval, 0.26-1.04;$P = 0.03$), but not the cortisol response (mean difference, -10.9 ng/ml, 95% confidence interval, -24.7 to 2.9;$P = 0.11$) in fetuses who had paired intrahepatic vein transfusions with and without fentanyl. Comparison with control fetuses transfused without fentanyl indicated that the β

endorphin and cerebral Doppler response to intrahepatic vein transfusion with fentanyl approached that of nonstressful placental cord transfusions.

"Conclusions: The authors conclude that intravenous fentanyl attenuates the fetal stress response to intrahepatic vein needling."

> Fisk NM, Gitau R, Teixeira MD, Giannakoulopoulos, X, Cameron, AD, Glover VA. Effect of Direct Fetal Opioid Analgesia on Fetal Hormonal and Hemodynamic Stress Response to Intrauterine Needling. *Anesthesiology*. 95 (2001) 828-835.

2. **De Buck, 2008**, p.294, col.2, para.4, "The autonomic and endocrine responses to noxious stimuli, the stress response, consist of the activation of the hypothalamic, pituitary, and adrenal axis.[15] Rises in blood levels of noradrenaline, cortisol and b-endorphin during invasive procedures in the human fetus are seen. Alterations in the brain blood flow have been seen as early as in the 18th week of pregnancy.[15] These autonomic effects of noxious stimulation can be suppressed by the administration of analgesics.[16]"

> De Buck F, Deprest J, Van de Velde M. Anesthesia for fetal surgery. *Current Opinion in Anaesthesiology*. 21 (2008) 293-297.
>
> [15]Rychik J, Tian Z, Cohen MS, Ewing SG, Cohen D, Howell LJ, Wilson RD, Johnson MP, Hedrick HL, Flake AW, Crombleholme TM, Adzick NS. Acute cardiovascular effects of fetal surgery in the human. *Circulation*. 110 (2004) 1549-1556.
>
> [16]Smith RP, Gitau R, Glover V, Fisk NM. Pain and stress in the human fetus. *European Journal of Obstetrics and Gynecology and Reproductive Biology*. 92 (2000) 161-165.

3. **Derbyshire, 2008**, p.119, col.2, para.1-2, "Anand's seminal work with neonates undergoing surgery demonstrated that fentanyl added to the anaesthetic regimen significantly reduces the stress response to invasive practice.[4] Specifically, plasma adrenalin, noradrenaline, glucagon, aldosterone, corticosterone, 11-deoxycorticosterone and 11-deoxycortisol levels were significantly increased in the nonfentanyl group up to 24 hours after surgery. Reducing the normal stress response was considered to be responsible for the improved clinical outcome of the fentanyl group who required less post-surgical ventilator support and had reduced circulatory and metabolic complications.

"More recently, the stress response to invasive practice has been examined in the fetus to demonstrate increased cortisol and h-endorphin circulation following intrauterine needling of the fetus beyond 18 weeks gestation.[25] Further studies have demonstrated that the fetal stress response includes haemodynamic changes in blood flow to protect essential organs, such as the brain, and blunting the stress response when providing opioid analgesia to the fetus.[26,27]"

> *Note: Derbyshire believes pain requires subjective human experience, not possible until after birth; nonetheless, he acknowledges this finding.*
>
> Derbyshire SW. Fetal Pain: Do We Know Enough to Do the Right Thing? *Reproductive Health Matters*. 16; 31Supp. (2008) 117-126.

[4] Anand KJ, Sippell WG, Aynsley-Green A. Randomised trial of fentanyl anaesthesia in preterm babies undergoing surgery: effects on the stress response. *Lancet.* 329 (1987) 62-66.

[25] Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling. *Lancet.* 344 (1994) 77-81

[26] Fisk NM, Gitau R, Teixeira MD, Giannakoulopoulos, X, Cameron, AD, Glover VA. Effect of Direct Fetal Opioid Analgesia on Fetal Hormonal and Hemodynamic Stress Response to Intrauterine Needling. *Anesthesiology.* 95 (2001) 828-835.

[27]Teixeira J, Fogliani R, Giannakoulopoulos X, Glover V, Fisk NM. Fetal haemodynamic stress response to invasive procedures. *Lancet.* 347 (1996) 624.

6: The position, asserted by some medical experts, that the unborn child is incapable of experiencing pain until a point later in pregnancy than 20 weeks after fertilization predominately rests on the assumption that the ability to experience pain depends on the cerebral cortex and requires nerve connections between the thalamus and the cortex. However, recent medical research and analysis, especially since 2007, provides strong evidence for the conclusion that a functioning cortex is not necessary to experience pain.

DOCUMENTATION:

a. **The position, asserted by some medical experts, that the unborn child is incapable of experiencing pain until a point later in pregnancy than 20 weeks after fertilization predominately rests on the assumption that the ability to experience pain depends on the cerebral cortex and requires nerve connections between the thalamus and the cortex.**

1. **Anand, 2006**, p.3, col.1, para.4 – col.2, para.2, "[R]ecent reviews purporting to rule out the occurrence of fetal pain.[3,4,22]... presuppose that cortical activation is necessary for fetal pain perception.[3,4,22] Based upon this assumption, the lack of evidence for pain-specific thalamocortical connections support their contention against fetal pain."

   Anand KJS. Fetal Pain? *Pain: Clinical Updates.* 14:2 (2006) 1-4.

   [3] Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

   [4] Mellor DJ, Diesch TJ, Gunn AJ, Bennet L. The importance of 'awareness' for understanding fetal pain. *Brain Research Reviews.* 49 (2005) 455-471.

   [22]Derbyshire SWG. Can fetuses feel pain? *British Medical Journal.* 332 (2006) 909-912.

2. **Royal College of Obstetricians & Gynecologists, 2010**, Summary, para.2, "In reviewing the neuroanatomical and physiological evidence in the fetus, it was apparent that connections from the periphery to the cortex are not intact before 24 weeks of gestation and, as most neuroscientists believe that the cortex is necessary for pain perception, it can be concluded that the fetus cannot experience pain in any sense prior to this gestation."

   Fetal Awareness: Review of Research and Recommendations for Practice. Report of a Working Party. *Royal College of Obstetricians and Gynecologists.* March 2010.

3. **Lee, 2005**, Abstract, para.3, "Pain perception requires conscious recognition or awareness of a noxious stimulus. Neither withdrawal reflexes nor hormonal stress response to invasive procedures prove the existence of fetal pain, because they can be elicited by nonpainful stimuli and occur without conscious cortical processing. Fetal awareness of noxious stimuli requires functional thalamocortical connections. Thalamocortical fibers begin appearing between 23 to 30 weeks' gestational age, while

eletroencephalography suggests the capacity for functional pain perception in preterm neonates probably does not exist before 29 or 30 weeks."

Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

4. **Brusseau, 2006**, p.190, col.2, para.4, "... such reflex responses to noxious stimuli have not been shown to involve the cortex and, thus, traditionally have not been thought to be available to conscious perception."

Brusseau R, Myers L. Developing consciousness: fetal anesthesia and analgesia. *Seminars in Anesthesia, Perioperative Medicine and Pain.* 25 (2006) 189-195.

5. **Mellor, 2005**, p.464, col.2, para.4, "[D]espite the presence of intact nociceptive pathways from around mid-gestation, the critical aspect of cortical awareness in the process of pain perception is missing."

Mellor DJ, Diesch TJ, Gunn AJ, Bennet L. The importance of 'awareness' for understanding fetal pain. *Brain Research Reviews.* 49 (2005) 455-471.

6. **Derbyshire, 2006**, p.910, col.1, para.2, "Current theories of pain consider an intact cortical system to be both necessary and sufficient for pain experience.[9,10]"

Derbyshire SWG. Can fetuses feel pain? *British Medical Journal.* 332 (2006) 909-912.

[9]Coghill RC, McHaffie JC, Yen YF. Neural correlates of interindividual difference in the subjective experience of pain. *Procedings of the National Academy of Science of the United States of America.* 100 (2003) 8538-8542.

[10]Derbyshire SWG, Whalley MG, Stenger VA, Oakley DA. Cerebral activation during hypnotically induced and imagined pain. *Neuroimage.* 23 (2004) 392-401.

b. However, recent medical research and analysis, especially since 2007, provides strong evidence for the conclusion that a functioning cortex is not necessary to experience pain.

1. **Merker, 2007**, p.80, col.2, para.3, "The evidence and functional arguments reviewed in this article are not easily reconciled with an exclusive identification of the cerebral cortex as the medium of conscious function... The tacit consensus concerning the cerebral cortex as the 'organ of consciousness' would thus have been reached prematurely, and may in fact be seriously in error."

Merker B. Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences.* 30 (2007) 63-81.

2. **Anand, 2007**, p.82, col.2, para.1, "A reappraisal of the mechanisms of huan consciousness, differentiating it from its attributes, functions, or contents, is long overdue. Widely held concepts about the key mechanisms of consciousness, or its fullest

expression via the human brain, have not been reexamined in the light of accumulating evidence since the 1970's. Merker presents the organization of a subcortical system…with multiple lines of anatomical, neurophysiological, behavioral, clinical, andneuropathological evidence, and a teleological rationale – all of which support a persuasive argument for the subcortical control and temporal sequencing of behavior.… One distressing impact of associating consciousness with cortical function, briefly mentioned by Merker in section 6 of the target article, pertains to the mistaken notions regarding pain perception in patient populations with impaired cortical function or cortical immaturity."

> Anand KJS. Consciousness, cortical function, and pain perception in nonverbal humans. *Behavioral and Brain Sciences*. 30:1 (2007) 82-83.

3. **Anand, 2006**, p.2, col.2, para.5, "Multiple lines of evidence thus corroborate that the key mechanisms of consciousness or conscious sensory perception are not dependent on cortical activity:"

> col.1, para.4, "Penfield and Jasper proposed that 'the highest integrative functions of the brain are not completed at the cortical level, but in a system of highly convergent subcortical structures supplying the key mechanism of consciousness.'"
>
> col.2, para.3, "Further clinical evidence for conscious perception mediated by subcortical centers comes from infants and children with hydranencephaly.[12,13]„
>
> col.2, para.4, "Thus, a subcortical system… mediates the organization of consciousness.[15]… That intact forebrain commissures are not required for high levels of cognitive function[16] provides further evidence for the subcortical integration…"
>
> "Whether consciousness is required for sensory perception has also been questioned by recent studies of adult patients in a persistent vegetative state.[17,18]„

p.3, col.1, para.4 – col.2, para.2, "[R]ecent reviews purporting to rule out the occurrence of fetal pain.[3,4,22]… presuppose that cortical activation is necessary for fetal pain perception.[3,4,22] Based upon this assumption, the lack of evidence for pain-specific thalamocortical connections support their contention against fetal pain. This line of reasoning, however, ignores clinical data cited above that ablation or stimulation of the primary somatosensory cortex does not alter pain perception in adults, whereas thalamic ablation or stimulation does. The thalamus plays a pivotal role in regulating the spinal-brainstem-spinal loops that mediate context-dependent descending facilitation or inhibition, coordinated via the key mechanisms underlying consciousness."

> Anand KJS. Fetal Pain? *Pain: Clinical Updates*. 14 (2006) 1-4.
>
> Penfield W, Jasper HH. *Epilepsy and the Functional Anatomy of the Human Brain*. Boston: Little, Brown & Co; 1954.

[3]Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

[4] Mellor DJ, Diesch TJ, Gunn AJ, Bennet L. The importance of 'awareness' for understanding fetal pain. *Brain Research Reviews.* 49 (2005) 455-471.

[12]Marin-Padilla M. Developmental neuropathology and impact of perinatal brain damage. *Journal of Neuropathology & Experimental Neurology.* 56 (1997) 219-235.

[13]Takada K, Shiota M, Ando M, et al. Porencephaly and hydranencephaly: a neuropathological study of four autopsy cases. *Brain Development.* 11 (1989) 51-56.

[14]Shewmon DA, Holmes GL, Byrne PA. Consciousness in congenitally decorticate children: Developmental vegetative state as self-fulfilling prophecy. *Developmental Medicine & Child Neurology.* 41 (1999) 364-374.

[15] Merker B. Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences.* 30 (2007) 63-81. [in press at time of citation by Anand]

[16]LeDoux JE, Risse GL, Springer SP, Wilson DH, Gazzaniga. Cognition and Commissurotomy. *Brain.* 100 (1997) 87-104.

[17]Shewmon DA. A critical analysis of conceptual domains of the vegetative state: sorting fact from fancy. *Neurorehabilitation.* 19 (2004) 364-374.

[18]Schiff NDM. *Neurology.* 64 (2005) 514-523.

[22]Derbyshire SWG. Can fetuses feel pain? *British Medical Journal.* 332 (2006) 909-912.

4. **Brusseau, 2008**, p.16, para.1, **"**However, if one were to argue that a minimal form of consciousness might be possible without cortical involvement, then certainly one would have to consider thalamic development as a benchmark for the possible generation of such a state. As described above, thalamic structures seem to be in place somewhere between 20 and 30 weeks… Other evidence, however, points to a much earlier maturation of thalamic processing function. Thalamic connections are intimately involved in the generation of the physiochemical and endocrine responses to nociception that are seen as early as 18 weeks.[20,27]"

   p.20, para.3, "Perhaps the subcortex is necessary and sufficient for at least a minimal, Hameroffian consciousness, one that (if the data regarding anencephalic children are to be believed) may render an integrated experience of nociception that we might call pain."

   Brusseau R. Developmental Perpectives: is the Fetus Conscious? *International Anesthesiology Clinics.* 46:3 (2008) 11-23.

   [20]Teixeira Jm, Glover V, Fisk NM. Acute cerebral redistribution in response to invasive procedure in the human fetus. *American Journal of Obstetrics & Gynecology.* 181 (1999) 1018-1025.

[27]Gitau R, Fisk NM, Teixeira JM, Cameron A, Glover V. Fetal hypothalamic–pituitary–adrenal stress responses to invasive procedures are independent of maternal responses. *Journal of Clinical Endocrinology and Metabolism.* 86 (2001) 104-109.

7: Substantial evidence indicates that children born missing the bulk of the cerebral cortex, those with hydranencephaly, nevertheless experience pain.

DOCUMENTATION:

1. **Brusseau, 2008**, p.17, para.2-3, "Clinical evidence for conscious perception mediated by such a subcortical system comes from infants and children with hydranencephaly…[31-33]. Despite the total or near-total absence of cerebral cortex, these children clearly demonstrate elements of consciousness.[34]… It is important to note that these are not hydrocephalic children who possess a thin rim of intact, functional cortex, but rather children with little or no cortex at all…what little cortex may remain is generally nonfunctional and without normal white matter connectivity.[35]

   "As such, it would seem these children demonstrate that anatomic development or functional activity of the cortex may not be required for conscious sensory perception. They may, and do in fact, respond to painful or pleasurable stimuli in what may easily be argued to be a conscious, coordinated manner, similar to intact children.[36]"

   Brusseau R. Developmental Perpectives: is the Fetus Conscious? *International Anesthesiology Clinics.* 46:3 (2008) 11-23.

   [31]Counter SA. Preservation of brainstem neurophysiological function in hydranencephaly. *Journal of Neuroscience*. 263 (2007) 198-207.

   [32]Marin-Padilla M. Developmental neuropathology and impact of perinatal brain damage. *Journal of Neuropathology & Experimental Neurology.* 56 (1997) 219-235.

   [33]Takada K, Shiota M, Ando M, et al. Porencephaly and hydranencephaly: a neuropathological study of four autopsy cases. *Brain Development.* 11 (1989) 51-56.

   [34]Shewmon DA, Holmes GL, Byrne PA. Consciousness in congenitally decorticate children: Developmental vegetative state as self-fulfilling prophecy. *Developmental Medicine & Child Neurology.* 41 (1999) 364-374.

   [35]Merker B. Life expectancy in hydranencephaly. *Clinical Neurology & Neurosurgery.* 110 (2008) 213-214.

   [36]McAbee GN, Chan A, Erde EL. Prolonged survival with hydranencephaly: report of two patients and literature review. *Pediatric Neurology.* 23 (2000) 80-84.

2. **Merker, 2007**, p.79, col.1, para.4, "My impression from this first-hand exposure to children with hydranencephaly confirms the account given by Shewmon and colleagues. These children are not only awake and often alert, but show responsiveness to their surroundings in the form of emotions or orienting reactions to environmental events… They express pleasure by smiling and laughter, and aversion by "fussing," arching of the back and crying (in many gradations), their faces being animated by these emotional states."

   Merker B. Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences.* 30 (2007) 63-81.

Shewmon DA, Holmes GL, Byrne PA. Consciousness in congenitally decorticate children: Developmental vegetative state as self-fulfilling prophecy. *Developmental Medicine & Child Neurology.* 41 (1999) 364-374.

3. **Brusseau, 2006**, p.191, col.1, para.1, "Indeed, there is evidence that hydranencephanic children responds to painful and pleasurable stimuli in a coordinated manner similar to other children.[11]"

   Brusseau R, Myers L. Developing consciousness: fetal anesthesia and analgesia. *Seminars in Anesthesia, Perioperative Medicine and Pain.* 25 (2006) 189-195.

   [11]Anand KJS. U.S. Congress. House of Representatives. Committee on the Judiciary. *Pain of the Unborn: Hearing Before the Subcommittee on the Constitution.* 109th Cong., 1st Sess., 2005.

4. **Beshkar, 2008**, p.554, col.1, para.1, "Shewmon et al. (1999) reported the cases of four children aged 5-17, with hydranencephaly involving complete or nearly complete absence of cerebral cortex. The authors observed that these children possessed a variety of cognitive capacities that were indicative of ordinary consciousness, including...appropriate affective responses."

   p.555, col.2, para.3, "Whether or not children born with hydranencephaly have consciousness is still controversial. However, the body of evidence in favor of the presence of consciousness in these patients seems to be more convincing than evidence and arguments against consciousness in such children."

   Beshker M. The Presence of Consciousness in the Absence of the Cerebral Cortex. *Synapse*. 62 (2008) 553-556.

   Shewmon DA, Holmes GL, Byrne PA. Consciousness in congenitally decorticate children: Developmental vegetative state as self-fulfilling prophecy. *Developmental Medicine & Child Neurology.* 41 (1999) 364-374.

8: In adults, stimulation or ablation of the cerebral cortex does not alter pain perception, while stimulation or ablation of the thalamus does.

DOCUMENTATION:

1. **Brusseau, 2008,** p.16, para.3, "In keeping with the critical insights of Penfield and Jasper, clinical evidence suggests that either ablation or stimulation of the primary somatosensory cortex does not alter pain perception in adults (demonstrated by Penfield and Jasper themselves), whereas both thalamic ablation and stimulation have been shown to interrupt pain perception."

   p.17, para.1 "In keeping with this evidence, we should consider that if cortical activity is not a prerequisite for pain perception in adults, then by analogy neither would it be a necessary criterion for fetuses."

   Note: Brusseau is ultimately agnostic regarding the ability of unborn children to feel pain before 28 weeks.

   Brusseau R. Developmental Perpectives: is the Fetus Conscious? *International Anesthesiology Clinics.* 46:3 (2008) 11-23.

   Penfield W, Jasper HH. *Epilepsy and the Functional Anatomy of the Human Brain.* Boston: Little, Brown & Co; 1954.

2. **Van Scheltema, 2008**, p.313, para.1, "Others however, argue that thalamocortical connections are not a necessary criterion for (fetal) pain perception as clinical data show that ablation or stimulation of the thalamus alone is sufficient to alter pain perception in adults.[11-14]"

   Van Scheltema PNA, Bakker S, Vandenbussche FPHA, Oepkes, D. Fetal Pain. *Fetal and Maternal Medicine Review*. 19:4 (2008) 311-324.

   [11]Brooks JK, Zambreanu L, Godinez A, Craig AD, Tracey I. Somatotopic organization of the human insula to painful heat studied with high resolution functional imaging. *Neuroimage*. 27 (2005) 201-209.

   [12]Craig AD. Interoception: the sense of the physiological condition of the body. *Current Opinion in Neurobiology*. 13 (2003) 500-505.

   [13]Nandi D, Aziz T, Carter H, Stein J. Thalamic field potentials in chronic central pain treated by periventricular gray stimulation – a series of eight cases. *Pain*. 101 (2003) 97-107.

   [14]Nandi D, Liu X, Joint C, Stein J, Aziz T. Thalamic field potentiasls during deep brain stimulation of periventricular gray in chronic pain. *Pain*. 97 (2002) 47-51.

3. **Merker, 2007**, p.65, col.1, para.3, "Penfield and Jasper note that cortical removal even as radical as hemispherectomy does not deprive a patient of consciousness, but rather of certain forms of information, discrimination capacities, or abilities, but not of

consciousness itself… What impressed Penfield and Jasper was the extent to which the cerebral cortex could be subjected to acute insult without producing so much as an interruption in the continuity of consciousness. Their opinion in this regard bears some weight, in that their magnum opus of 1954 – *Epilepsy and the Functional Anatomy of the Human Brain* summarizes and evaluates experience with 750 such operations."

Merker B. Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences.* 30 (2007) 63-81.

Penfield W, Jasper HH. *Epilepsy and the Functional Anatomy of the Human Brain.* Boston: Little, Brown & Co; 1954.

4. **Morsella, 2010**, p.15, col.1, para.3, "It seems that consciousness can persist even when great quantities of the cortex are absent."

Morsella E, Krieger SC, Bargh JA. Minimal neuroanatomy for a conscious brain: Homing in on the networks constituting consciousness. *Neural Networks*. 23 (2010) 14-15.

9: **Substantial evidence indicates that structures used for pain processing in early development differ from those of adults, using different neural elements available at specific times during development, such as the subcortical plate, to fulfill the role of pain processing.**

DOCUMENTATION:

1. **Anand, 2006**, p.3, col.1, para.5, "Clinical and animal research shows that the fetus or neonate is not a 'little adult,' that the structures used for pain processing in early development are unique and different from those of adults, and that many of these fetal structures and mechanisms are not maintained beyond specific periods of early development. The immature pain system thus uses the neural elements available during each stage of development to carry out its signaling role."

   Anand KJS. Fetal Pain? *Pain: Clinical Updates.* 14:2 (2006) 1-4.

2. **Van Sheltema, 2008**, p.313, para.1; "[P]ain perception during fetal and neonatal development does not necessarily involve the same structures involved in pain processing as those in adults, meaning that the lack of development of certain connections is not sufficient to support the argument that fetuses can not feel pain until late gestation.[10] Some say even that the structures used for pain processing in the fetus are completely different from those used by adults and that many of these structures are not maintained beyond specific periods of early development.[8,15]"

   Van Scheltema PNA, Bakker S, Vandenbussche FPHA, Oepkes, D. Fetal Pain. *Fetal and Maternal Medicine Review*. 19:4 (2008) 311-324.

   [10] Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

   [8]Fitzgerald M. The Development of Nociceptive Circuits. *Nature Reviews: Neuroscience.* 6 (2005) 507-520.

   [15]White, MC, Wolf, AR. Pain and Stress in the Human Fetus. *Best Practice & Research Clinical Anaesthesiology.* 18 (2004) 205-220.

3. **White, 2004**, p.208, para.4, "The anatomical evidence shows that the nociceptive connections of the fetus are not merely immature versions of the adult but are structurally different and these differences confer differences in function. Furthermore, interference with the natural progression to adult-like status can have extensive effects. Nerve section of afferent pathways, from the forelimb in the rat during early development, results in major changes in the subsequent central connections and sensory perception from other sites.[40] Clearly this has implications for any form of fetal surgery."

   White, MC, Wolf, AR. Pain and Stress in the Human Fetus. *Best Practice & Research Clinical Anaesthesiology.* 18 (2004) 205-220.

[40]Killackey HP & Dawson DR. Expansion of the central hindpaw representation following fetal forelimb removal in the rat. *European Journal of Neuroscience* 1 (1989) 210-221.

4. **Fitzgerald, 2005**, p.507, col.1, para.2, "Newborn infants show strong pain behaviour, but the study of the development of nociceptive pathways shows that their pain involves functional signaling pathways that are not found in the mature nervous system in healthy individuals."

Fitzgerald M. "The Development of Nociceptive Circuits."*Nature Reviews: Neuroscience.* 6 (2005) 507-520.

10: The position, asserted by some medical experts, that the unborn child remains in a coma-like sleep state that precludes the unborn child experiencing pain is inconsistent with the documented reaction of unborn children to painful stimuli and with the experience of fetal surgeons who have found it necessary to sedate the unborn child with anesthesia to prevent the unborn child from thrashing about in reaction to invasive surgery.

DOCUMENTATION:

a. The position, asserted by some medical experts, that the unborn child remains in a coma-like sleep state that precludes the unborn child experiencing pain…

1. **Royal College of Obstetricians & Gynecologists, 2010**, Summary, para.2, "Furthermore, there is increasing evidence that the fetus never experiences a state of true wakefulness *in utero* and is kept, by the presence of its chemical environment, in a continuous sleep-like unconsciousness or sedation."

   Fetal Awareness: Review of Research and Recommendations for Practice. Report of a Working Party. *Royal College of Obstetricians and Gynecologists.* March 2010.

2. **Fitzgerald, 2005**, p.513, col.1, para.2, "Despite the existence of sensory reflexes from the first trimester of human fetal life, it is unlikely that the fetus is ever awake or aware and, therefore, able to truly experience pain, due to high levels of endogenous neuroinhibitors, such as adenosine and pregnanolone, which are produced in the feto-placental unit and contribute to fetal sleep states[144]. In preterm infants below 32 weeks most pain responses, including facial expressions, seem to be largely subcortical[145]."

   Fitzgerald M. The Development of Nociceptive Circuits. *Nature Reviews: Neuroscience.* 6 (2005) 507-520.

3. **Mellor, 2005**, p.464, col.2, para.4, "We conclude that there is currently no strong evidence to suggest that the fetus is ever awake, even transiently; rather, it is actively kept asleep (and unconscious) by a variety of endogenous inhibitory factors. Thus, despite the presence of intact nociceptive pathways from around mid-gestation, the critical aspect of cortical awareness in the process of pain perception is missing."

   Mellor DJ, Diesch TJ, Gunn AJ, Bennet L. The importance of 'awareness' for understanding fetal pain. *Brain Research Reviews.* 49 (2005) 455-471.

b. … is inconsistent with the documented reaction of unborn children to painful stimuli and with the experience of fetal surgeons who have found it necessary to sedate the unborn child with anesthesia to prevent the unborn child from thrashing about in reaction to invasive surgery.

1. **Van de Velde, 2005**, p.256, col.2, para.2, "In our trial inadvertent touching of an immobilized fetus resulted in fetal 'awakening.'"

   Van de Velde M, Van Schoubroeck DV, Lewi LE, Marcus MAE, Jani JC, Missant C, Teunkens A, Deprest J. Remifentanil for Fetal Immobilization and Maternal Sedation During Fetoscopic Surgery: A Randomized, Double-Blind Comparison with Diazepam. *Anesthesia & Analgesia*. 101 (2005) 251-258.

2. **Giannakoulopoulos, 1994**, p.77, col.2, para.3, "We have observed that the fetus reacts to intrahepatic vein needling with vigorous body and breathing movements, which are not present during placental cord insertion needling."

   Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and β-endorphin response to intrauterine needling. *Lancet*. 344 (1994) 77-81.

3. **Lee, 2005**, p.951, col.1, para.3, "…they [fetal anesthesia and analgesia] serve other purposes unrelated to pain reduction, including (1) inhibiting fetal movement during a procedure.[63-65]"

   Note: Lee et al. believe that pain is an emotional and psychological experience, possible only after 29-30 weeks gestation. Nonetheless, they recognize the necessity of immobilizing the unborn child during surgery before this point due to coordinated movements in response to invasive procedures.

   Lee SJ, Ralston HJP, Drey EA, Partridge, JC, Rosen, MA. A Systematic Multidisciplinary Review of the Evidence. *Journal of the American Medical Association.* 294:8 (2005) 947-954.

   [63]Seeds JW, Corke BC, Spielman FJ. "Prevention of fetal movement during invasive procedures with pancuronium bromide." *American Journal of Obstetetrics & Gynecology*. 155 (1986) 818-819.

   [64]Rosen MA. Anesthesia for fetal procedures and surgery. *Yonsei Medical Journal*. 42 (2001) 669-680.

   [65]Cauldwell CB. Anesthesia for fetal surgery. *Anesthesiology Clinics of North America*. 20 (2002) 211-226.

4. **Van Scheltema, 2008**, p.319, para.2, "Besides the argument of achieving adequate fetal anaesthesia, there are other purposes that justify the administration of drugs: the inhibiting fetal movement during a procedure…[15,67-72]"

   Van Scheltema PNA, Bakker S, Vandenbussche FPHA, Oepkes, D. Fetal Pain. *Fetal and Maternal Medicine Review*. 19:4 (2008) 311-324.

   [15] White, MC, Wolf, AR. Pain and Stress in the Human Fetus. *Best Practice & Research Clinical Anaesthesiology*. 18 (2004) 205-220.

   [67] Seeds JW, Corke BC, Spielman FJ. Prevention of fetal movement during invasive procedures with pancuronium bromide. *American Journal of Obstetetrics & Gynecology*. 155 (1986) 818-819.

[68]Rosen MA. Anesthesia for procedures involving the fetus. *Seminars in Perinatology.* 12 (1991) 410-417.

[69] Rosen MA. Anesthesia for fetal procedures and surgery. *Yonsei Medical Journal.* 42 (2001) 669-680.

[70]Cauldwell CB. Anesthesia for fetal surgery. *Anesthesiology Clinics of North America.* 20 (2000) 211-226.

[71]Smith RP, Gitau R, Glover V, Fisk NM. Pain and stress in the human fetus. *European Journal of Obstetrics and Gynecology and Reproductive Biology.* 92 (2000) 161-165.

[72]Schwarz U, Galinkin JL. Anesthesia for fetal surgery. *Seminars on Pediatric Surgery.* 12 (2003) 196-201.

11: Consequently, there is substantial medical evidence that an unborn child is capable of experiencing pain by 20 weeks after fertilization.

DOCUMENTATION:

1. **Wright, 2005**, p.26, para.8 – p.27, para.3, "After 20 weeks of gestation, an unborn child has all the prerequisite anatomy, physiology, hormones, neurotransmitters, and electrical current to "close the loop" and create the conditions needed to perceive pain…The development of the perception of pain beings at the 6th week of life. By 20 weeks, and perhaps even earlier, all the essential components of anatomy, physiology, and neurobiology exist to transmit painful sensations from the skin to the spinal cord and to the brain.*"

   **From the testimony of Dr. Jean A. Wright, Professor And Chair of Pediatrics, Mercer School of Medicine*

   U.S. Congress. House of Representatives. Committee on the Judiciary. *Pain of the Unborn: Hearing Before the Subcommittee on the Constitution.* 109th Cong., 1st Sess., 2005.

2. **Anand, 2005**, p.38, "My opinion is, based on evidence suggesting that the types of stimulation that will occur during abortion procedures, very likely most fetuses at 20 weeks after conception will be able to perceive that as painful, unpleasant, noxious stimulation.*"

   **From the testimony of Dr. Sunny Anand, Director, Pain Neurobiology Laboratory, Arkansas Children's Hospital Research Institute, and Professor of Pediatrics, Anesthesiology, Pharmacology, and Neurobiology, University of Arkansas College of Medicine*

   U.S. Congress. House of Representatives. Committee on the Judiciary. *Pain of the Unborn: Hearing Before the Subcommittee on the Constitution.* 109th Cong., 1st Sess., 2005.

3. **Anand, 2006**, p.3, col.2, "Our current understanding of development provides the anatomical structures, the physiological mechanisms, and the functional evidence for pain perception developing in the second trimester, certainly not in the first trimester, but well before the third trimester of human gestation."

   Anand KJS. Fetal Pain? *Pain: Clinical Updates.* 14:2 (2006) 1-4.

4. **Glover, 1999**, p.885, col.1, para.3, "Given the anatomical evidence, it is possible that the fetus can feel pain from 20 weeks and is caused distress by interventions from as early as 15 or 16 weeks."

   Glover V. Fetal pain: implications for research and practice. *British Journal of Obstetrics and Gynaecology.* 106 (1999) 881-886.

5. **Gibbins, 2007**, p.224, col.2, para.1, "Current data suggest that by 26 and even as early as 20 weeks gestation, a rudimentary pain pathway may be present."

> Gibbins S, Golec L. "It Will Not Hurt a Bit," "What You Do Not Know Cannot Hurt You," and Other Myths About Fetal Surgical Pain. *Newborn & Infant Nursing Reviews.* 7:4 (2007) 224-226.

6. **Brusseau, 2006**, p.191, col.2, para.1, "In fact there are thought to be transient cholinergic neurons with functioning synapses connecting the thalamus and cortical plate from approximately 20 weeks. This time point could be taken as the absolute earliest time in gestation when a fetus could be aware of nociceptive stimuli, or to 'feel pain.'"

> Brusseau R, Myers L. Developing consciousness: fetal anesthesia and analgesia. *Seminars in Anesthesia, Perioperative Medicine and Pain.* 25 (2006) 189-195.

7. **Van Scheltema, 2008**, p.320, para.3, "Neuroanatomical, neurophysiological, hormonal, haemodynamic and behavioural data indicate that a fetus is capable of reacting to noxious stimuli, implying that the fetus can experience stress and possibly even pain…It is difficult to determine from what gestation onwards fetal anaesthesia should be provided; however, we feel that it should be considered from at least mid-gestation."

> Van Scheltema PNA, Bakker S, Vandenbussche FPHA, Oepkes, D. Fetal Pain. *Fetal and Maternal Medicine Review*. 19:4 (2008) 311-324.

Mr. FRANKS. This bill regulates all forms of late-term abortions, each of them gruesome and painful. Babies are dismembered, or they are chemically burned alive through saline abortion. Some late-term abortions kill the child in utero through lethal injection before removing the child, and this can be done with the physician

puncturing the small, pain-cable baby through the chest to inject drugs that will end the child's life.

Most Americans think that late-term abortions are rare, but, in fact, they make up about 10 percent of abortions annually. With an average of greater than 1.2 million abortions in the U.S. each year, that comes to approximately 120,000 late-term abortions annually, or greater than 325 late-term abortions every day in America.

H.R. 3803 is long overdue, and it is a law which protects unborn children who have reached 20 weeks development from abortions on the basis that the unborn child feels pain by at least this stage of development, if not much earlier. The bill provides an exception where an abortion is necessary to save the life of the mother.

When a pregnancy endangers the mother's life, there are only two options: abortion, or delivery. Due to medical advancements it is now nearly always possible to deliver the baby in under half an hour through emergency C-section rather than through a late-term abortion, which typically requires hours or even days to complete. Delivery by C-section is generally substantially faster and, therefore, more safe for the mother and the child where the pregnancy results or presents an imminent threat to life.

With this in mind, H.R. 3803 provides that the physician must choose the option that is most likely to save the life of both patients, mother and baby. Currently there are no restrictions on abortions clear up until the moment of birth in the District of Columbia other than the Federal law that bans partial-birth abortions, a law that passed by the U.S. Congress and not the D.C. government some years ago.

Many Americans are unaware that the unborn child feels pain, and certainly most people believe that they can trust the medical profession to know if the child does and to administer anesthesia as a basic requirement of human compassion. But, in fact, there is no standard legal rule to provide that an unborn child receive anesthesia. This is true whether the child is a wanted child that is undergoing surgery in utero, or whether the child is an unwanted child or other child that is undergoing an abortion. In this respect unborn children receive less legal protection from completely unnecessary cruelty than farm animals, which have protection under the Human Slaughter Act.

This is barbaric, ladies and gentlemen, and we must not allow it to happen in America. We must enact protections for unborn children to put an end to this, the greatest human rights violation occurring on U.S. soil, the painful late-term abortion that has already victimized potentially millions of pain-cable unborn Americans since the Supreme Court gave America abortion on demand in 1973.

And with that, I would yield to the Ranking Member of the Subcommittee Mr. Nadler for his opening statement.

The bill, H.R. 3803, follows:]

I

112TH CONGRESS
2D SESSION

H. R. 3803

To amend title 18, United States Code, to protect pain-capable unborn children in the District of Columbia, and for other purposes.

IN THE HOUSE OF REPRESENTATIVES

JANUARY 23, 2012

Mr. FRANKS of Arizona (for himself, Mr. AKIN, Mr. GOHMERT, Mr. FLEMING, Mr. WALBERG, Mr. HUELSKAMP, Mr. PITTS, Mr. LAMBORN, Mr. SMITH of Texas, Mr. KINGSTON, Mr. SMITH of New Jersey, Mr. SOUTHERLAND, Mrs. SCHMIDT, Mr. ADERHOLT, Mr. HARRIS, Mr. BUCSHON, Mr. PENCE, Mr. HULTGREN, Mr. BOUSTANY, Mr. ROGERS of Alabama, Mr. MANZULLO, Mr. ROSS of Florida, Mrs. HARTZLER, Mr. FORTENBERRY, Mr. HERGER, Mr. CANSECO, Mr. LANKFORD, Mrs. LUMMIS, Mr. AUSTIN SCOTT of Georgia, Mr. ROE of Tennessee, Mr. NUNNELEE, Mr. MARCHANT, Mr. HUIZENGA of Michigan, Mr. MURPHY of Pennsylvania, Mr. JONES, Mr. LANDRY, Mr. BACHUS, Mr. ROGERS of Kentucky, Mrs. ROBY, Mr. MCKINLEY, Mr. LIPINSKI, Mr. KELLY, Mr. GOWDY, Mr. JORDAN, Mrs. BACHMANN, Mrs. ELLMERS, Mr. AMASH, Mr. ISSA, Mr. SCHWEIKERT, and Mr. SCALISE) introduced the following bill; which was referred to the Committee on the Judiciary, and in addition to the Committee on Oversight and Government Reform, for a period to be subsequently determined by the Speaker, in each case for consideration of such provisions as fall within the jurisdiction of the committee concerned

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A BILL

To amend title 18, United States Code, to protect pain-capable unborn children in the District of Columbia, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

This Act may be cited as the "District of Columbia Pain-Capable Unborn Child Protection Act".

SEC. 2. LEGISLATIVE FINDINGS.

Congress finds and declares the following:

(1) Pain receptors (nociceptors) are present throughout the unborn child's entire body and nerves link these receptors to the brain's thalamus and subcortical plate by no later than 20 weeks after fertilization.

(2) By 8 weeks after fertilization, the unborn child reacts to touch. After 20 weeks, the unborn child reacts to stimuli that would be recognized as painful if applied to an adult human, for example, by recoiling.

(3) In the unborn child, application of such painful stimuli is associated with significant increases in stress hormones known as the stress response.

(4) Subjection to such painful stimuli is associated with long-term harmful neurodevelopmental effects, such as altered pain sensitivity and, possibly, emotional, behavioral, and learning disabilities later in life.

(5) For the purposes of surgery on unborn children, fetal anesthesia is routinely administered and

is associated with a decrease in stress hormones compared to their level when painful stimuli are applied without such anesthesia.

(6) The position, asserted by some medical experts, that the unborn child is incapable of experiencing pain until a point later in pregnancy than 20 weeks after fertilization predominately rests on the assumption that the ability to experience pain depends on the cerebral cortex and requires nerve connections between the thalamus and the cortex. However, recent medical research and analysis, especially since 2007, provides strong evidence for the conclusion that a functioning cortex is not necessary to experience pain.

(7) Substantial evidence indicates that children born missing the bulk of the cerebral cortex, those with hydranencephaly, nevertheless experience pain.

(8) In adult humans and in animals, stimulation or ablation of the cerebral cortex does not alter pain perception, while stimulation or ablation of the thalamus does.

(9) Substantial evidence indicates that structures used for pain processing in early development differ from those of adults, using different neural elements available at specific times during develop-

ment, such as the subcortical plate, to fulfill the role of pain processing.

(10) The position, asserted by some commentators, that the unborn child remains in a coma-like sleep state that precludes the unborn child experiencing pain is inconsistent with the documented reaction of unborn children to painful stimuli and with the experience of fetal surgeons who have found it necessary to sedate the unborn child with anesthesia to prevent the unborn child from engaging in vigorous movement in reaction to invasive surgery.

(11) Consequently, there is substantial medical evidence that an unborn child is capable of experiencing pain at least by 20 weeks after fertilization, if not earlier.

(12) It is the purpose of the Congress to assert a compelling governmental interest in protecting the lives of unborn children from the stage at which substantial medical evidence indicates that they are capable of feeling pain.

(13) The compelling governmental interest in protecting the lives of unborn children from the stage at which substantial medical evidence indicates that they are capable of feeling pain is intended to be separate from and independent of the compelling

governmental interest in protecting the lives of unborn children from the stage of viability, and neither governmental interest is intended to replace the other.

(14) The District Council of the District of Columbia, operating under authority delegated by Congress, repealed all limitations on abortion at any stage of pregnancy, effective April 29, 2004.

(15) Article I, section 8 of the Constitution of the United States of America provides that the Congress shall "exercise exclusive Legislation in all Cases whatsoever" over the District established as the seat of government of the United States, now known as the District of Columbia. The constitutional responsibility for the protection of pain-capable unborn children within the Federal District resides with the Congress.

SEC. 3. DISTRICT OF COLUMBIA PAIN-CAPABLE UNBORN CHILD PROTECTION.

(a) IN GENERAL.—Chapter 74 of title 18, United States Code, is amended by inserting after section 1531 the following:

"§ 1532. District of Columbia pain-capable unborn child protection

"(a) UNLAWFUL CONDUCT.—Notwithstanding any other provision of law, including any legislation of the District of Columbia under authority delegated by Congress, it shall be unlawful for any person to perform an abortion within the District of Columbia, or attempt to do so, unless in conformity with the requirements set forth in subsection (b).

"(b) REQUIREMENTS FOR ABORTIONS.—

"(1) The physician performing or attempting the abortion shall first make a determination of the probable post-fertilization age of the unborn child or reasonably rely upon such a determination made by another physician. In making such a determination, the physician shall make such inquiries of the pregnant woman and perform or cause to be performed such medical examinations and tests as a reasonably prudent physician, knowledgeable about the case and the medical conditions involved, would consider necessary to make an accurate determination of post-fertilization age.

"(2)(A) Except as provided in subparagraph (B), the abortion shall not be performed or attempted, if the probable post-fertilization age, as de-

termined under paragraph (1), of the unborn child is 20 weeks or greater.

"(B) Subject to subparagraph (C), subparagraph (A) does not apply if, in reasonable medical judgment, the abortion is necessary to save the life of a pregnant woman whose life is endangered by a physical disorder, physical illness, or physical injury, including a life-endangering physical condition caused by or arising from the pregnancy itself, but not including psychological or emotional conditions or any claim or diagnosis that the woman will engage in conduct which she intends to result in her death.

"(C) A physician terminating or attempting to terminate a pregnancy under the exception provided by subparagraph (B) may do so only in the manner which, in reasonable medical judgment, provides the best opportunity for the unborn child to survive, unless, in reasonable medical judgment, termination of the pregnancy in that manner would pose a greater risk of—

"(i) the death of the pregnant woman; or

"(ii) the substantial and irreversible physical impairment of a major bodily function, not

including psychological or emotional conditions, of the pregnant woman;

than would other available methods.

"(c) CRIMINAL PENALTY.—Whoever violates subsection (a) shall be fined under this title or imprisoned for not more than 2 years, or both.

"(d) BAR TO PROSECUTION.—A woman upon whom an abortion in violation of subsection (a) is performed or attempted may not be prosecuted under, or for a conspiracy to violate, subsection (a), or for an offense under section 2, 3, or 4 based on such a violation.

"(e) CIVIL REMEDIES.—

"(1) CIVIL ACTION BY WOMAN ON WHOM THE ABORTION IS PERFORMED.—A woman upon whom an abortion has been performed or attempted in violation of subsection (a), may in a civil action against any person who engaged in the violation obtain appropriate relief.

"(2) CIVIL ACTION BY RELATIVES.—The father of an unborn child who is the subject of an abortion performed or attempted in violation of subsection (a), or a maternal grandparent of the unborn child if the pregnant woman is an unemancipated minor, may in a civil action against any person who engaged in the violation, obtain appropriate relief, un-

less the pregnancy resulted from the plaintiff's criminal conduct or the plaintiff consented to the abortion.

"(3) APPROPRIATE RELIEF.—Appropriate relief in a civil action under this subsection includes—

"(A) objectively verifiable money damages for all injuries, psychological and physical, occasioned by the violation of this section;

"(B) statutory damages equal to three times the cost of the abortion; and

"(C) punitive damages.

"(4) INJUNCTIVE RELIEF.—

"(A) IN GENERAL.—A qualified plaintiff may in a civil action obtain injunctive relief to prevent an abortion provider from performing or attempting further abortions in violation of this section.

"(B) DEFINITION.—In this paragraph the term 'qualified plaintiff' means—

"(i) a woman upon whom an abortion is performed or attempted in violation of this section;

"(ii) any person who is the spouse, parent, sibling or guardian of, or a current

or former licensed health care provider of, that woman; or

"(iii) the United States Attorney for the District of Columbia.

"(5) ATTORNEYS FEES FOR PLAINTIFF.—The court shall award a reasonable attorney's fee as part of the costs to a prevailing plaintiff in a civil action under this subsection.

"(6) ATTORNEYS FEES FOR DEFENDANT.—If a defendant in a civil action under this section prevails and the court finds that the plaintiff's suit was frivolous and brought in bad faith, the court shall also render judgment for a reasonable attorney's fee in favor of the defendant against the plaintiff.

"(7) AWARDS AGAINST WOMAN.—Except under paragraph (6), in a civil action under this subsection, no damages, attorney's fee or other monetary relief may be assessed against the woman upon whom the abortion was performed or attempted.

"(f) PROTECTION OF PRIVACY IN COURT PROCEEDINGS.—

"(1) IN GENERAL.—Except to the extent the Constitution or other similarly compelling reason requires, in every civil or criminal action under this section, the court shall make such orders as are nec-

essary to protect the anonymity of any woman upon whom an abortion has been performed or attempted if she does not give her written consent to such disclosure. Such orders may be made upon motion, but shall be made sua sponte if not otherwise sought by a party.

"(2) ORDERS TO PARTIES, WITNESSES, AND COUNSEL.—The court shall issue appropriate orders under paragraph (1) to the parties, witnesses, and counsel and shall direct the sealing of the record and exclusion of individuals from courtrooms or hearing rooms to the extent necessary to safeguard her identity from public disclosure. Each such order shall be accompanied by specific written findings explaining why the anonymity of the woman must be preserved from public disclosure, why the order is essential to that end, how the order is narrowly tailored to serve that interest, and why no reasonable less restrictive alternative exists.

"(3) PSEUDONYM REQUIRED.—In the absence of written consent of the woman upon whom an abortion has been performed or attempted, any party, other than a public official, who brings an action under paragraphs (1), (2), or (4) of subsection (e) shall do so under a pseudonym.

"(4) LIMITATION.—This subsection shall not be construed to conceal the identity of the plaintiff or of witnesses from the defendant or from attorneys for the defendant.

"(g) REPORTING.—

"(1) DUTY TO REPORT.—Any physician who performs or attempts an abortion within the District of Columbia shall report that abortion to the relevant District of Columbia health agency (hereinafter in this section referred to as the 'health agency') on a schedule and in accordance with forms and regulations prescribed by the health agency.

"(2) CONTENTS OF REPORT.—The report shall include the following:

"(A) POST-FERTILIZATION AGE.—For the determination of probable postfertilization age of the unborn child, whether ultrasound was employed in making the determination, and the week of probable post-fertilization age that was determined.

"(B) METHOD OF ABORTION.—Which of the following methods or combination of methods was employed:

"(i) Dilation, dismemberment, and evacuation of fetal parts also known as 'dilation and evacuation'.

"(ii) Intra-amniotic instillation of saline, urea, or other substance (specify substance) to kill the unborn child, followed by induction of labor.

"(iii) Intracardiac or other intra-fetal injection of digoxin, potassium chloride, or other substance (specify substance) intended to kill the unborn child, followed by induction of labor.

"(iv) Partial-birth abortion, as defined in section 1531.

"(v) Manual vacuum aspiration without other methods.

"(vi) Electrical vacuum aspiration without other methods.

"(vii) Abortion induced by use of mifepristone in combination with misoprostol; or

"(viii) if none of the methods described in the other clauses of this subparagraph was employed, whatever method was employed.

"(C) AGE OF WOMAN.—The age or approximate age of the pregnant woman.

"(D) COMPLIANCE WITH REQUIREMENTS FOR EXCEPTION.—The facts relied upon and the basis for any determinations required to establish compliance with the requirements for the exception provided by subsection (b)(2).

"(3) EXCLUSIONS FROM REPORTS.—

"(A) A report required under this subsection shall not contain the name or the address of the woman whose pregnancy was terminated, nor shall the report contain any other information identifying the woman.

"(B) Such report shall contain a unique Medical Record Number, to enable matching the report to the woman's medical records.

"(C) Such reports shall be maintained in strict confidence by the health agency, shall not be available for public inspection, and shall not be made available except—

"(i) to the United States Attorney for the District of Columbia or that Attorney's delegate for a criminal investigation or a civil investigation of conduct that may violate this section; or

"(ii) pursuant to court order in an action under subsection (e).

"(4) PUBLIC REPORT.—Not later than June 30 of each year beginning after the date of enactment of this paragraph, the health agency shall issue a public report providing statistics for the previous calendar year compiled from all of the reports made to the health agency under this subsection for that year for each of the items listed in paragraph (2). The report shall also provide the statistics for all previous calendar years during which this section was in effect, adjusted to reflect any additional information from late or corrected reports. The health agency shall take care to ensure that none of the information included in the public reports could reasonably lead to the identification of any pregnant woman upon whom an abortion was performed or attempted.

"(5) FAILURE TO SUBMIT REPORT.—

"(A) LATE FEE.—Any physician who fails to submit a report not later than 30 days after the date that report is due shall be subject to a late fee of $1,000 for each additional 30-day period or portion of a 30-day period the report is overdue.

"(B) COURT ORDER TO COMPLY.—A court of competent jurisdiction may, in a civil action commenced by the health agency, direct any physician whose report under this subsection is still not filed as required, or is incomplete, more than 180 days after the date the report was due, to comply with the requirements of this section under penalty of civil contempt.

"(C) DISCIPLINARY ACTION.—Intentional or reckless failure by any physician to comply with any requirement of this subsection, other than late filing of a report, constitutes sufficient cause for any disciplinary sanction which the Health Professional Licensing Administration of the District of Columbia determines is appropriate, including suspension or revocation of any license granted by the Administration.

"(6) FORMS AND REGULATIONS.—Not later than 90 days after the date of the enactment of this section, the health agency shall prescribe forms and regulations to assist in compliance with this subsection.

"(7) EFFECTIVE DATE OF REQUIREMENT.—Paragraph (1) of this subsection takes effect with respect to all abortions performed on and after the

first day of the first calendar month beginning after the effective date of such forms and regulations.

"(h) DEFINITIONS.—In this section the following definitions apply:

"(1) ABORTION.—The term 'abortion' means the use or prescription of any instrument, medicine, drug, or any other substance or device—

"(A) to intentionally kill the unborn child of a woman known to be pregnant; or

"(B) to otherwise intentionally terminate the pregnancy of a woman known to be pregnant with an intention other than to increase the probability of a live birth, to preserve the life or health of the child after live birth, or to remove a dead unborn child who died as the result of natural causes in utero, accidental trauma, or a criminal assault on the pregnant woman or her unborn child, and which causes the premature termination of the pregnancy.

"(2) ATTEMPT AN ABORTION.—The term 'attempt', with respect to an abortion, means conduct that, under the circumstances as the actor believes them to be, constitutes a substantial step in a course of conduct planned to culminate in performing an abortion in the District of Columbia.

"(3) FERTILIZATION.—The term 'fertilization' means the fusion of human spermatozoon with a human ovum.

"(4) HEALTH AGENCY.—The term 'health agency' means the Department of Health of the District of Columbia or any successor agency responsible for the regulation of medical practice.

"(5) PERFORM.—The term 'perform', with respect to an abortion, includes induce an abortion through a medical or chemical intervention including writing a prescription for a drug or device intended to result in an abortion.

"(6) PHYSICIAN.—The term 'physician' means a person licensed to practice medicine and surgery or osteopathic medicine and surgery, or otherwise licensed to legally perform an abortion.

"(7) POST-FERTILIZATION AGE.—The term 'post-fertilization age' means the age of the unborn child as calculated from the fusion of a human spermatozoon with a human ovum.

"(8) PROBABLE POST-FERTILIZATION AGE OF THE UNBORN CHILD.—The term 'probable post-fertilization age of the unborn child' means what, in reasonable medical judgment, will with reasonable probability be the postfertilization age of the unborn

child at the time the abortion is planned to be performed or induced.

"(9) REASONABLE MEDICAL JUDGMENT.—The term 'reasonable medical judgment' means a medical judgment that would be made by a reasonably prudent physician, knowledgeable about the case and the treatment possibilities with respect to the medical conditions involved.

"(10) UNBORN CHILD.—The term 'unborn child' means an individual organism of the species homo sapiens, beginning at fertilization, until the point of being born alive as defined in section 8(b) of title 1.

"(11) UNEMANCIPATED MINOR.—The term 'unemancipated minor' means a minor who is subject to the control, authority, and supervision of a parent or guardian, as determined under the law of the State in which the minor resides.

"(12) WOMAN.—The term 'woman' means a female human being whether or not she has reached the age of majority.".

(b) CLERICAL AMENDMENT.—The table of sections at the beginning of chapter 74 of title 18, United States Code, is amended by adding at the end the following new item:

"1532. District of Columbia pain-capable unborn child protection.".

(c) CHAPTER HEADING AMENDMENTS.—

(1) CHAPTER HEADING IN CHAPTER.—The chapter heading for chapter 74 of title 18, United States Code, is amended by striking "**PARTIAL BIRTH ABORTIONS**" and inserting "**ABORTIONS**".

(2) TABLE OF CHAPTERS FOR PART I.—The item relating to chapter 74 in the table of chapters at the beginning of part I of title 18, United States Code, is amended by striking "**PARTIAL BIRTH ABORTIONS**" and inserting "**ABORTIONS**".

○

Mr. NADLER. Thank you, Mr. Chairman.

We are back again considering legislation that would curtail women's reproductive rights. I understand how personally important this is to some of my colleagues, and they are certainly entitled to their beliefs, but the many Americans who see the world very differently, including millions of women who value their personal autonomy and their personal liberty, can be forgiven if this looks just like another battle in the Republican war on women.

I accept that on this one we are going to have to agree to disagree. In this case my colleagues appear, through the operation of the criminal code, to be trying to settle a scientific question on which there is no consensus within the field. That is an exercise of raw political power, not a dispassionate fact-finding. And, of course, the exercise of political power doesn't alter scientific fact.

Some of the views we are going to hear today are, in fact, viewed by many in the field as outliers, not as mainstream scientific thought. The fact that the majority has allowed three individuals to purport to represent this as clearly established science, views that are clearly a marginal view in the scientific community, will create a false and misleading record.

The fact that the minority has been limited to one witness only demonstrates just what a farce these hearings are. Yes, I know we could have invited our own medical and scientific expert, but that would have been at the expense of hearing from an actual woman who can provide a real-world look at the impact this legislation will have on real families.

I know we could have invited the Delegate from the District of Columbia, the only Member of this body elected to represent the only Americans who would be directly affected by this bill, but that would have to be at the expense of hearing either from a person with real experience in this area, or from a medical expert and a scientific expert with more mainstream views. The exclusion of Delegate Norton, who is relegated to sitting in the audience today—and I want to welcome her and apologize for the rudeness my Republican colleagues are showing a colleague by refusing her request to be heard—is yet another example of that abuse of power.

Yes, the Constitution gives Congress plenary power over the District, something that we can and should remedy, and have remedied to some extent in the District of Columbia Governance Act, but are ignoring today, but just because we have the power to impose our will on people who have no voice does not make it right or moral.

As I have said in the past, never in my 20 years as a Member of this body have I seen a colleague treated so contemptuously. The gentlewoman from the District of Columbia is a Member of this body, and the people she represents are taxpaying American citizens who serve in our military; respond when one of us has an emergency requiring police, fire, or EMT services; and serve as congressional staff, without whom we could not do our work. And yet this Committee cannot be bothered to take 5 minutes to hear our colleague who will not be permitted to vote on this bill.

The District of Columbia is not a colony, it is part of the United States, and its people are entitled to be treated with the same respect that we demand for the people we represent, and it is uncon-

scionable that she is not permitted to testify other than as the one minority witness.

I ask unanimous consent to place the gentlewoman's statement in the record.

Mr. FRANKS. Without objection.

[The prepared statement of Ms. Norton follows:]

ELEANOR HOLMES NORTON
DISTRICT OF COLUMBIA

COMMITTEE ON TRANSPORTATION AND INFRASTRUCTURE
SUBCOMMITTEES
RANKING MEMBER, ECONOMIC DEVELOPMENT, PUBLIC BUILDINGS AND EMERGENCY MANAGEMENT
AVIATION
WATER RESOURCES AND ENVIRONMENT

COMMITTEE ON OVERSIGHT AND GOVERNMENT REFORM
SUBCOMMITTEES
HEALTH CARE, DISTRICT OF COLUMBIA, CENSUS AND THE NATIONAL ARCHIVES
FEDERAL WORKFORCE, U.S. POSTAL SERVICE AND LABOR POLICY
GOVERNMENT ORGANIZATION, EFFICIENCY AND FINANCIAL MANAGEMENT

Congress of the United States
House of Representatives
Washington, DC 20515–1501

STATEMENT OF
CONGRESSWOMAN ELEANOR HOLMES NORTON
OF THE DISTRICT OF COLUMBIA
ON H.R. 3803, THE DISTRICT OF COLUMBIA PAIN-CAPABLE UNBORN CHILD PROTECTION ACT
HOUSE COMMITTEE ON THE JUDICIARY, SUBCOMMITTEE ON THE CONSTITUTION
MAY 17, 2012

What matters in the submission of this testimony is what H.R. 3803 and this subcommittee are attempting to do to the citizens I represent, and, therefore, I submit this testimony as part of my responsibility to them, and ask that it be included in the record of today's hearing. However, my constituents would also count on me to note for the record the subcommittee's callous disregard of long-standing congressional courtesy in denying my request to testify, in addition to the invited witnesses, particularly considering that the subject matter under consideration affects only my district. Unlike every member of this subcommittee, I am elected by, and am accountable to, the residents of the District of Columbia.

This is the second time in the 112th Congress that the majority has focused exclusively on my district while denying my request to testify. How very easy it is for the majority to gang up on the District of Columbia after supporting the continuing denial of its tax-paying citizens to representation in the House and Senate. How irresistible it has been to pick on the District of Columbia and its citizens with not one but two bills that the majority dares not try to apply to all citizens of the United States. The lack of courage of the majority's convictions is breathtaking. Common courtesy and the congressional tradition of comity and respect demand that the Member elected to speak for the only Americans affected by a bill be allowed to speak for them, regardless of other witnesses who may speak to the underlying issue. Last year, I was denied to speak on H.R. 3, a bill that would permanently prohibit only one jurisdiction, the District of Columbia, from spending its local funds on abortions for low-income women. Today it is H.R. 3803, which would bar the women of only one district, the District of Columbia, from having abortions after 20 weeks of pregnancy. Fortunately, the majority has not yet found a way to completely silence our residents. I thank the minority for inviting Professor Christy Zink, who has agreed to speak for us, as few others could, as a mother whose tragic experience compelled an abortion after 20 weeks into her pregnancy.

Some are debating whether Republicans have been engaging in a "war on women" in our country. What is not debatable is the Republican fixation on the women of the District of Columbia. The Republican majority, which was elected on a promise of jobs and devolving power to state and local governments, brought the federal government (and with it, the District of Columbia government) to within an hour of shutting down in April 2011, and relented only after

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it succeeded in re-imposing an undemocratic rider on a spending bill that prohibits the District of Columbia from spending its own local funds on abortions for low-income women. Although the abortion rider remains in place today, it has not satisfied the apparently insatiable hunger of Republicans to expand the reach of the federal government into local affairs. Today, they are moving from interfering with the decisions of low-income women in the District of Columbia, to attacking every woman in the District of Columbia.

H.R. 3803 is unprincipled twice over. It is the first bill ever introduced in Congress that would deny constitutional rights to the citizens of only one jurisdiction in the United States, and it is the first bill ever introduced in Congress that would ban abortions after twenty weeks of pregnancy. Republicans claim that the bill does not usurp local authority because Congress has jurisdiction over the District of Columbia. However, that argument has been unavailing for 39 years, since Congress gave up that power over the District of Columbia, except for a small number of enumerated exceptions, with passage of the Home Rule Act of 1973. The right to reproductive choice was not among those exceptions.

The supporters of H.R. 3803 surely know that it is unconstitutional on two counts. The bill violates the reproductive rights spelled out in *Roe v. Wade*, as well as the 14th Amendment right to equal treatment under the law by intentionally discriminating against women who live in the nation's capital. D.C. residents are used to Members piling on, but we will never hesitate to fight back, especially when Members have the audacity to try to place our citizens outside the protections of the U.S. Constitution, as H.R. 3803 does. As the Supreme Court said in *Callan v. Wilson*, "There is nothing in the history of the Constitution or of the original amendments to justify the assertion that the people of th[e] District [of Columbia] may be lawfully deprived of the benefit of any of the constitutional guarantees of life, liberty, and property."

Why, then, a hearing today on a bill that violates the right to reproductive freedom, equal protection, and federalism all at once? The answers are inescapable. Republicans do not dare take on the women of this country who have voting Members of the House and Senate with a post-20-week ban on abortions. Instead, the majority has chosen a cheap and cynical way to make its ideological point during an election year. With last year's civil disobedience, D.C. residents and officials showed that we will never accept second-class treatment of our city. Today we want this subcommittee to know that we will never accept second-class treatment of our citizens, either.

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Mr. NADLER. Thank you.

I am not going to sit here and debate the question of fetal pain, except to note that even Dr. Anand, who is cited in the majority witness testimony and hearing memo and was called by the majority to testify before this Subcommittee in 2005, told us, and I quote, "I think the evidence for and against fetal pain is very uncertain at the present time. There is consensus in the medical and scientific research community that there is no possibility of pain or

pain perception in the first trimester. There is uncertainty in the second trimester," unquote.

The Journal of the American Medical Association concluded that, quote, "Evidence regarding the capacity for fetal pain is limited, but indicates that fetal perception of pain is unlikely before the third trimester."

The Royal Academy of Obstetricians and Gynecologists concluded, quote, "It can be concluded that the fetus cannot experience pain in any sense prior to 24 weeks gestation," closed quote.

Are we really going to take sides in this scientific debate by jailing and bankrupting people who don't agree, or actually agree with the majority of the scientific community? Because that is about what this bill would do. Similarly, the claim that abortion is never necessary to protect the woman's health is simply not one that is widely held in the medical profession, and the idea that we should be enshrining these marginal views into the criminal code defies reason.

There are many difficult issues that we should deal with and deal with in a more serious and exhaustive manner, but I guess if you have the votes, and the Constitution gives you imperial powers, what the heck.

And one additional problem with this bill: The bill is facially unconstitutional. The Supreme Court has told us in many cases that we have no authority to ban abortion in the second trimester; e.g., 20 weeks. And we have no authority to ban abortion without a health exception, not just the life exception for the mother, which this bill does.

I find it deeply disturbing that when it comes to issues like this, some people think there is nothing wrong with making families in crisis have the courage of legislators' convictions. That is just wrong. We hear a lot of rhetoric about freedom, but here we are telling women they have no freedom to make their own decisions; we will make their decisions for them because we know the morality, we know the right, we know the religion, and to heck with what they think, and to heck with what they believe, and to heck with what their religion tells them. That is wrong.

Mr. Chairman, I yield back the balance of my time.

Mr. FRANKS. Thank you, Mr. Nadler.

Mr. FRANKS. Let me, before we begin, comment briefly on the issue of Delegate Norton. Per our usual procedures, the Republicans are allowed to invite three witnesses to the hearing, and the Democrats are allowed to invite one. This is not a departure. When the Democrats were in charge, this is exactly the proportion that was always used.

The Ranking Member has complete discretion regarding whom the Democrats witnesses will be, and in this case the Ranking Member chose Ms. Zink. We do not have a tradition, policy, or practice of deviating from our normal practice of allowing the minority a proportionate number of witness invitations. Ranking Member Nadler had the opportunity to invite one witness to this hearing. He chose Ms. Zink, a resident of Washington, D.C. He had every opportunity to invite Delegate Norton as his witness. He chose not to.

But any written submission by Delegate Norton will, of course, be made part of the hearing record per our usual procedures, and we welcome her contributions, and I would certainly invite Delegate Norton to sit on the dais here with us. Our Committee policy prevents noncommittee members from being recognized for any purpose, but she is certainly welcome to sit with us, and I extend that invitation with every goodwill in my heart.

Mr. NADLER. Mr. Chairman?

Mr. FRANKS. With that, Ms. Norton, would you like to sit on the dais with us?

Ms. NORTON. Thank you, no.

Mr. FRANKS. All right. I understand.

So I thank the gentleman.

Mr. NADLER. A point of clarification.

Mr. FRANKS. Sure.

Mr. NADLER. Mr. Chairman, the Ranking Member—I, of course, had the right to pick one delegate—one witness. However, when we were in—when the Democrats were in charge, and frankly on other Committees today, when a colleague wishes to testify, that colleague is afforded a separate panel, or colleagues are afforded a separate panel, and is not counted as the one witness for the minority. We had a choice.

Mr. FRANKS. I am going to require the time back here. The reality——

Mr. NADLER. I would like to finish my statement on this.

Mr. FRANKS. All right.

Mr. NADLER. We had a choice. It is wrong to impose a choice on us when legislation affects a specific district. If this were the Transportation Committee, and we were having a debate over a bridge in Oshkosh, we would, of course, invite the Representative from Oshkosh to testify, and that wouldn't count against in the normal panel. And that had been our practice. It was our practice in the past. It ought to be the practice. It is disrespectful to the District otherwise.

Mr. FRANKS. The gentleman knows that every piece of legislation affects many different Members of this Congress. If we were to follow the gentleman's suggestion, the room would be full of Members of Congress. And I would just suggest that the gentleman knows that there is no deviation from any rules that we have had previous to today. This is exactly the same rules as always. And the gentleman knows that, and I am afraid that we are approaching an effort to change the subject here. The gentleman said he did not wish to debate pain for the unborn child, and that is indeed the subject of this hearing.

So I thank the gentleman and the Ranking Member of the full Committee. Let us see, we don't have anyone else.

So we are going to move on to witness introductions right now. And I would introduce first Dr. Anthony Levatino. Am I saying that right? He is a board-certified obstetrician/gynecologist. In his 32-year career, he has practiced obstetrics and gynecology in both private and university settings, including as an associate professor of an OB-GYN—of OB-GYN at Albany Medical College.

Thank you for being here, sir.

Dr. Colleen Malloy, or Malloy?

Dr. MALLOY. Malloy.

Mr. FRANKS. Malloy—serves as assistant professor in the division of neonatology in the Department of Pediatrics at Northwestern University Feinberg School of Medicine.

Dr. Byron Calhoun serves as a professor and vice chair of the department of obstetrics and gynecology at West Virginia University, Charleston. Dr. Calhoun has a specialty in caring for high-risk pregnancies.

Thank you for being here, Dr. Calhoun.

Our final witness, is Christy Zink, a resident of Washington, D.C. And thank you for being here, Christy.

I thank all of the witnesses for appearing before us today. Each of the witnesses' written statements will be entered into the record in its entirety.

I ask that each witness summarize his or her testimony in 5 minutes or less, and to help you stay within that time, there is a timing light on your table. When the light switches from green to yellow, you will have 1 minute to conclude your testimony. When the light turns red, it signals that the witness' 5-minutes have expired.

And before I recognize the witnesses, it is the tradition of this Subcommittee that they be sworn. So if you will please stand to be sworn.

[Witnesses sworn.]

Mr. FRANKS. Thank you. Please be seated.

Also, the witnesses, please turn your microphone on before speaking. We have a lot of fun with that.

And I would now recognize our first witness Mr. Levatino—Dr. Levatino for 5 minutes.

TESTIMONY OF ANTHONY LEVATINO, M.D., OBSTETRICS AND GYNECOLOGY

Dr. LEVATINO. Chairman Franks and distinguished Members of the Subcommittee, my name is Anthony Levatino. I am a board-certified obstetrician/gynecologist. I received my medical degree from Albany Medical College in Albany, New York, in 1976, and completed my OB-GYN residency at Albany Medical Center in 1980. Over my 32-year career, I have been privileged to practice obstetrics and gynecology in both private and university settings, and from June 1993 until September 2000, I was an associate professor of OB-GYN at Albany Medical College, serving at different times as the medical student director and residency program director. I have also been in private practice and currently operate a solo gynecology practice in Las Cruces, New Mexico.

Thank you for the invitation to address this issue.

During my residency training during the first—and during my first 5 years of private practice, I performed both first- and second-trimester abortions. During my residency years, second-trimester abortions were typically performed using saline infusions or occasionally prostaglandin instillation techniques. These procedures were difficult, expensive, and necessitated the patients go through labor to expel their preborn children.

By 1980, at the time I entered private practice first in Florida and then in upstate New York, those of us in the abortion industry

were looking for a more efficient method of second-trimester abortion. We found that suction dilatation evacuation, or suction D&E for short, offered clear advantages over the older instillation methods. The procedure was much quicker and never ran the risk of a live birth.

Understand that my partner and I were not running an abortion clinic. We practiced general obstetrics and gynecology, but abortion was definitely a part of our practice. Relatively few gynecologists in upstate New York would perform such a procedure at the time, and we saw an opportunity to expand our abortion practice. I performed first-trimester suction dilatation and curettage abortions in my office up to 10 weeks from last menstrual period and later procedures in an outpatient hospital setting.

From 1981 through February 1985, I performed approximately 1,200 abortions. Over 100 of them were second-trimester D&E procedures up to 24 weeks of gestation from last menstrual period, equivalent to 22 weeks postfertilization age.

As an aside, the last menstrual period dating system and postfertilization dating systems are equally valid, and both are found in the practice of medicine and in mainstream medical literature. Most, if not all, embryology textbooks, for example, typically date fetal development in terms of days or week postfertilization. In clinical obstetrics we use the last menstrual period system. Both are valid. It is only necessary that one specify which system is utilized, and H.R. 3803 does that. Any competent physician can read the definitions in H.R. 3803 and understand exactly where that cut-off line is.

Imagine, if you can, that you are a prochoice obstetrician/gynecologist like I was. Your patient today is 24 weeks pregnant, measured last menstrual period as obstetricians typically do. At 24 weeks from last menstrual period, her uterus is two finger breadths above her umbilicus. If you could see her baby, which would be easy on an ultrasound, that baby would be as your hand plus a half from head to rump, not counting the legs.

Your patient has been feeling her baby kick for the last month or more, and now she is asleep on an operating room table, and you are there to help her with her problem pregnancy. The first task is to remove the laminaria that had earlier been placed in the cervix, the opening to the uterus, to dilate it sufficiently to allow the procedure that you are about to perform.

With that accomplished, direct your attention to the surgical instruments arranged on the right. The first instrument you will need is a 14 French suction catheter. I brought one along so you don't have to imagine it. It is about 9 inches long. It is clear plastic, and there is an opening through the center of it.

Picture yourself, if you can, taking this instrument and introducing it through the cervix, and instructing your circulating nurse to turn on the suction machine. What you will see is pale yellow fluid running through this through the tubing into the suction machine. That was the amniotic fluid that was there originally to protect the baby.

You are next going to need a Sopher clamp. It is about 13 inches long, it is stainless steel, and the jaw on this is composed of rows of sharp teeth. You introduce this instrument blindly and start

pulling off limbs. Feel yourself grabbing and pulling hard, and I do mean hard, and out pops an arm about that long, which you put down next to you. Follow that by a leg, just as long, and then you tear out the intestine, the spine, heart and lungs.

The difficult part of the procedure is the head, which is about the size of a plum. You know you have got it right if you—again, this is blind—but you know you have got it right if your instrument is spread about as far as it can go. And you have got ahold of this, and you know you did it right if you crush down and a white material runs out of the cervix. That was the baby's brains. Then you will pull out scull pieces. Many times a little face will come back and stare back at you.

Congratulations. You have just successfully performed a D&E abortion. And if you think that doesn't hurt, if you believe that that isn't an agony for this child, please think again.

Mr. FRANKS. Thank you, Dr. Levatino.

[The prepared statement of Dr. Levatino follows:]

Testimony of Anthony Levatino, MD, JD

before the Subcommittee on the Constitution,

Committee on the Judiciary,

U.S. House of Representatives

on The District of Columbia Pain-Capable Unborn Child Protection Act (H.R. 3803)

May 17, 2012

Chairman Franks and distinguished members of the subcommittee, my name is Anthony Levatino. I am a board-certified obstetrician gynecologist. I received my medical degree from Albany Medical College in Albany, New York in 1976, and completed my OB-GYN residency training at Albany Medical Center in 1980. In my 32-year career, I have been privileged to practice obstetrics and gynecology in both private and university settings. From June 1993 until September 2000, I was associate professor of OB-GYN at the Albany Medical College, serving at different times as both medical student director and residency program director. I have also dedicated many years to private practice and currently operate a solo gynecology practice in Las Cruces, New Mexico. I appreciate your kind invitation to address issues related to the District of Columbia Pain-Capable Unborn Child Protection Act (H.R. 3803).

During my residency training and during my first five years of private practice, I performed both first and second-trimester abortions. During my residency years, second-trimester abortions were typically performed using saline infusion or, occasionally, prostaglandin instillation techniques. These procedures were difficult, expensive and necessitated that patients go through labor to expel their pre-born children. By 1980, at the time I entered private practice first in Florida and then in upstate New York, those of us in the abortion industry were looking for a more efficient method of second-trimester abortion. We found that the "Suction dilation and evacuation" procedure (or "Suction D&E") offered clear advantages over older installation methods. The procedure was much quicker and never ran the risk of a live birth.

Understand that my partner and I were not running an abortion clinic. We practiced general obstetrics and gynecology, but abortion was definitely part of that practice. Relatively few gynecologists in upstate New York would perform such a procedure at the time, and we saw an opportunity to expand our abortion practice. I performed first-trimester suction dilation and curettage abortions in my office up to 10 weeks from last menstrual period and later procedures in an outpatient hospital setting. From 1981 through February 1985, I performed approximately 1200 abortions. Over 100 of them were second-trimester Suction D&E procedures up to 24 weeks gestation, by which I mean 24 weeks from the first day of the woman's last menstrual period (LMP), which is equivalent to 22 weeks post-fertilization age.

As an aside, both the LMP dating system and the post-fertilization dating system are equally valid and both are found in the practice of medicine and in mainstream medical literature. Most if not all embryology textbooks, for example, typically date fetal development in terms of days or weeks post-fertilization. In clinical obstetrics we use the LMP system. Both are perfectly valid. It is only necessary that one specify which system is being utilized, and H.R. 3803 does that. Any competent physician can read the definitions in H.R. 3803 and understand exactly where the cut off line is.

Imagine, if you can, that you are a pro-choice obstetrician/gynecologist like I once was. Your patient today is 24 weeks pregnant (LMP). At twenty-four weeks from last menstrual period, her uterus is two finger-breadths above the umbilicus. If you could see her baby, which is quite easy on an ultrasound, she would be as long as your hand plus a half, from the top of her head to the bottom of her rump, not counting the legs. Your patient has been feeling her baby kick for the last month or more, but now she is asleep on an operating room table and you are there to help her with her problem pregnancy.

The first task is to remove the laminaria that had earlier been placed in the cervix, the opening to the uterus, to dilate it sufficiently to allow the procedure you are about to perform. With that accomplished, direct your attention to the surgical instruments arranged on a small table to your right. The first instrument you reach for is a 14-French suction catheter. It is clear plastic and about nine inches long. It has a bore through the center approximately ¾ of an inch in diameter. Picture yourself introducing this catheter through the cervix and instructing the circulating nurse to turn on the suction machine, which is connected through clear plastic tubing to the catheter. What you will see is a pale yellow fluid the looks a lot like urine coming through the catheter into a glass bottle on the suction machine. This is the amniotic fluid that surrounded the baby to protect her.

With suction complete, look for your Sopher clamp. This instrument is about thirteen inches long and made of stainless steel. At the business end are located jaws about 2 inches long and about 1/2 an inch wide with rows of sharp ridges or teeth. This instrument is for grasping and crushing tissue. When it gets hold of something, it does not let go. A second trimester D&E abortion is a blind procedure. The baby can be in any orientation or position inside the uterus. Picture yourself reaching in with the Sopher clamp and grasping anything you can. At twenty-four weeks gestation, the uterus is thin and soft so be careful not to perforate or puncture the walls. Once you have grasped something inside, squeeze on the clamp to set the jaws and pull hard – really hard. You feel something let go and out pops a fully formed leg about six inches long. Reach in again and grasp whatever you can. Set the jaw and pull really hard once again and out pops an arm about the same length. Reach in again and again with that clamp and tear out the spine, intestines, heart and lungs.

The toughest part of a D&E abortion is extracting the baby's head. The head of a baby that age is about the size of a large plum and is now free floating inside the uterine cavity. You can be pretty sure you have hold of it if the Sopher clamp is spread about as far as your fingers will allow. You know you have it right when you crush down on the clamp and see white gelatinous material coming through the cervix. That was the baby's brains. You can then extract

the skull pieces. Many times a little face may come out and stare back at you. Congratulations! You have just successfully performed a second-trimester Suction D&E abortion.

If you refuse to believe that this procedure inflicts severe pain on that unborn child, please think again.

Before I close, I want to make a comment on the claims that I often hear that we must keep abortion legal in order to save women's lives, or prevent grave physical health damage, in cases of acute conditions that can and do arise in pregnancy. Albany Medical Center, where I worked for over seven years, is a tertiary referral center that accepts patients with life-threatening conditions related to or caused by pregnancy. I personally treated hundreds of women with such conditions in my tenure there. There are several conditions that can arise or worsen, typically during the late second or third trimester of pregnancy, that require immediate care. In many of those cases, ending or "terminating" the pregnancy, if you prefer, can be life saving, but "terminating a pregnancy" does not necessarily mean "abortion." I maintain that abortion is seldom if ever a useful intervention in these cases.

Here is why: Before a Suction D&E procedure can be performed, the cervix must first be sufficiently dilated. In my practice, this was accomplished with serial placement of laminaria. Laminaria is a type of sterilized seaweed that absorbs water over several hours and swells to several times its original diameter. Multiple placements of several laminaria at a time are absolutely required prior to attempting a suction D&E. In the mid-second trimester, this requires approximately 36 hours to accomplish. If one were to use the alternate method defined in federal law as Partial-Birth Abortion (but now generally banned), this process requires three days, as explained by Dr. Martin Haskell in his 1992 paper that first described this type of abortion.

In cases where a pregnancy places a woman in danger of death or grave physical injury, a doctor more often than not doesn't have 36 hours, much less 72 hours, to resolve the problem. Let me illustrate with a real-life case that I managed while at the Albany Medical Center. A patient arrived one night at 28 weeks gestation with severe pre-eclampsia or toxemia. Her blood pressure on admission was 220/160. A normal blood pressure is approximately 120/80. This patient's pregnancy was a threat to her life and the life of her unborn child. She could very well be minutes or hours away from a major stroke. This case was managed successfully by rapidly stabilizing the patient's blood pressure and "terminating" her pregnancy by Cesarean section. She and her baby did well. This is a typical case in the world of high-risk obstetrics. In most such cases, any attempt to perform an abortion "to save the mother's life" would entail undue and dangerous delay in providing appropriate, truly life-saving care. During my time at Albany Medical Center I managed hundreds of such cases by "terminating" pregnancies to save mother's lives. In all those cases, the number of unborn children that I had to deliberately kill was zero.

Mr. FRANKS. Dr. Malloy, you are recognized now for 5 minutes.

TESTIMONY OF COLLEEN A. MALLOY, M.D., ASSISTANT PROFESSOR, DIVISION OF NEONATOLOGY/DEPARTMENT OF PEDIATRICS, NORTHWESTERN UNIVERSITY FEINBERG SCHOOL OF MEDICINE

Dr. MALLOY. I am here today to talk to you as a neonatologist about fetal pain.

We have gone over the dating systems. It is very important to differentiate between the postfertilization age and the last menstrual period dating. I am here because it is easy for me to imagine these babies at 20 or 24 weeks postfertilization age because they are my patients in the NICU.

So at 21 postfertilization age, for example, it is a 53 percent survival to discharge to home, published in June of 2009. This is another example of a chart showing the survival to discharge in Pediatrics 2010: Postfertilization age at 20 weeks, only 6 percent; 21 weeks, 25 percent; and at 22 weeks, over half of those babies survive to go home. And our hospital data is very similar. The 22- to 24-week post-fertilization age data, 80 percent of those babies discharge to home.

So these are some pictures of what the babies look like in utero 14 weeks post-fertilization through 22 weeks postfertilization. You can see the detail in the face. You can see the movements that 4-D ultrasounds that we have now are realtime images. The baby is kicking, moving, sucking their thumb, doing all things babies do in a smaller state. A picture of a 20-week postfertilization baby here, and these are my patients. This is that same infant when they are born and when we take care of them every day in our NICU.

This is a 22-week postfertilization baby. Very common, 24-week LMP baby in our NICU. We take care of these babies all the time. They survive, they do well, and go home.

This baby is 25 weeks by LMP. Survival rate is upwards of 85 percent. When we have a 25-week baby at our NICU, the assumption is the baby will do well, go home with mom.

So when you look at the milestones of pain development, it happens early on. Eight weeks face and skin receptors appear. Fourteen weeks, the sensory fibers grow into the spinal cord. By 15 weeks the monoamine fibers reach the cortex, and by 20 weeks all the pain receptors are present and linked. The cerebral cortex, at 20 weeks the fetal brain actually has a full complement of neurons that are present in adulthood. At 20 weeks you can do EEG recordings on the babies. At 22 weeks we do EEGs on our patients, and they have the same EEG patterns that you see in a neonate born at term.

There is behavioral responses as evidence for pain. At 8 weeks the fetus makes movements. Again, we have 4-D ultrasound that shows 3-D images of babies kicking, moving, practically dancing in the womb. At 20 weeks the fetus responds to sound, and many studies' published literature have shown that they react to stimuli by moving away from painful stimuli, by wincing, recoiling, vigorous body movements. You can see it in realtime. It is like watching a movie.

There have been studies that look at the fetus when you can sample blood through the baby's liver versus sampling blood through the umbilical cord, and there is no neurons and no nerve tissues that the baby would sense pain from the umbilical cord, but when you take blood from a baby's liver, it feels it. It moves away from the needle, and the stress hormones of the baby, which are measurable, go up by 500 percent.

So the hormonal response to pain in these babies, which I see every day, are identical between the fetus, the premature baby, and even the adult. The stress hormone response for a premature infant, again, rises upwards of 500 percent. The cortisol, which is the same hormone that we can measure in adults, is approximately 200 percent increased. And this is beginning at 18 weeks gestation we can measure this, and have measured this and published it.

When you look at neuropeptides and pain, the neuropeptides that help populate the signal for pain, substance P and enkephalin, I found very early, 11 weeks and 13 weeks.

There is actually published data showing that it is the later part of the pregnancy in which the descending inhibitory pathways of fetal pain develop, meaning that the first part of pregnancy is actually when the pain system develops, and the latter part is when the pain mitigating systems develop. So actually, some people believe the fetuses feel more pain than later-born infants. And the evidence that supports that is that increased concentrations of drugs are required for sedation of premature infants.

Again, the stress hormone response is actually higher in premature infants than adults undergoing similar surgeries, such as cardiac surgery. The pain transmitters in the spine are abundant, and the pain-inhibiting transmitters that we all have are sparse in the premature infant.

So again, if you look at this slide, here is the pain system developing, here is the gestation in weeks, and the pain modifying system really doesn't happen until later on. So they are basically just a raw bundle of nerves in the NICU. And these are the patients that I perform procedures on every day, and I can guarantee you that when I put a chest tube in, or I intubate a patient, or I put an IV in, they feel it.

This is actually a picture of a woman I had the privilege of meeting who was born 23 years ago. At that time she was the smallest surviving premie. She was 24 weeks postfertilization age. She weighed 280 grams, less than a Coke can. And she went on to be an honor student in college.

That same hospital in 2004 actually broke their own record. This baby was 25 weeks LMP, weighed 244 grams, and is now doing well in elementary school. She has a twin sister, and they are both actually doing very well.

So in my experience as a neonatologist, I would just like to mention that it is no longer a mystery what is going on in the womb, because those same babies come to me, and I see them firsthand every day and work with their families and, we can see how they react to pain when we do procedures in the NICU.

One of the most basic of government principles is that the State should protect its members from harm. Technology, imaging and clinical neonatology enable us to know much more about fetal life

than ever before. We now understand the fetus to be a developing, moving, interacting member of the human family who feels pain, just as we feel pain. If we are to be a benevolent society, we are bound to protect the fetus. We should not tolerate the gruesome and painful procedures being performed on the smallest of our Nation.

Thank you.

Mr. FRANKS. Thank you, Dr. Malloy.

[The prepared statement of Dr. Malloy follows:]

Testimony of Colleen A. Malloy, MD
Assistant Professor, Division of Neonatology/ Department of Pediatrics
Northwestern University Feinberg School of Medicine
Before the Subcommittee on the Constitution,
Committee on the Judiciary,
U.S. House of Representatives

May 17, 2012

Chairman Franks and distinguished members of the subcommittee, my name is Colleen A. Malloy. I serve as an assistant professor in the Division of Neonatology in the Department of Pediatrics at Northwestern University Feinberg School of Medicine. Thank you for this opportunity to testify regarding some of the scientific and clinical issues that are pertinent to your consideration of the District of Columbia Pain-Capable Unborn Child Protection Act (H.R. 3803).

This legislation would prohibit abortion within the District of Columbia, a federal jurisdiction, beginning at 20 weeks fetal age. This age is equivalent to 22 weeks in the "LMP" system of dating, which is commonly used in obstetrics and neonatology. The bill contains an exception for certain cases in which an abortion is deemed necessary because a grave physical condition endangers the mother's life.

With the advancement of in utero imaging, blood sampling, and fetal surgery, we now have a much better understanding of life in the womb than we did at the time that *Roe v. Wade* was handed down. Our generation is the beneficiary of new information which allows us to understand more thoroughly the existence and importance of fetal and neonatal pain. As noted in my biography, I am trained and board-certified in the field of neonatology. The standard of care in my field recognizes neonatal pain as an important entity to be acknowledged, measured, and treated.

With advancements in neonatology and perinatal medicine, we have been able to push back the age at which a neonate can be resuscitated and resuscitated successfully. When we speak of infants at 22 weeks LMP, for example, we no longer have to rely solely on inferences or ultrasound imagery, because such premature patients are kicking, moving, reacting, and developing right before our eyes in the Neonatal Intensive Care Unit.

In neonatology, we describe the age of neonates in terms of the last menstrual period (LMP) dating system, which dates a pregnancy starting with day zero as the first day of the last menstrual period. However, the actual development in the womb is commonly referred to with post-fertilization dating. This bill utilizes the post-fertilization system of dating. These approaches are equally valid, as long as one remembers which dating system is being employed in any particular discussion. The LMP age is the post-fertilization age, plus two weeks. Thus, the cutoff point in this legislation is 20 weeks after fertilization, which would be 22 weeks in the LMP system. In today's medical arena, we resuscitate patients at this age and are able to witness their ex-utero growth and development.

Medical advancement and technology have enabled us to improve our ability to care for these infants. In June 2009, the *Journal of American Medical Association* reported a Swedish series of over 300,000 infants. Survival to 1 year of life of live born infants at 20, 21, 22, 23, and 24 weeks post-fertilization age was 10%, 53%, 67%, 82%, and 85%, respectively. In September 2010, *Pediatrics* reported survival to discharge rates of 9575 infants at a number of academic institutions in the US.

The results were similar, with survival at 20, 21, 22, 23, and 24 weeks post-fertilization age being 6%, 26%, 55%, 72%, and 84%, respectively. As we provide care for all these survivors, we are able to witness their experiences with pain. In fact, standard of care for neonatal intensive care units requires attention to and treatment of neonatal pain. There is no reason to believe that a born infant would feel pain any differently than that same infant were he or she still in utero. Thus, the difference between fetal and neonatal pain is simply the locale in which the pain occurs. The receiver's experience of the pain is the same. I could never imagine subjecting my tiny patients to horrific procedures such as those that involve limb detachment or cardiac injection.

There is ample biologic, physiologic, hormonal, and behavioral evidence for fetal and neonatal pain. As early as 8 weeks post-fertilization, face skin receptors appear. At 14 weeks, sensory fibers grow into the spinal cord and connect with the thalamus. At 13-16 weeks, monoamine fibers reach the cerebral cortex, so that by 17-20 weeks the thalamo-cortical relays penetrate the cortex. Many authors have substantiated that pain receptors are present and linked by no later than 20 weeks post-fertilization. (Myers 2004; Derbyshire 2010; Anand 1987; Vanhalto 2000; Brusseau 2008; VanScheltema 2008). In fact, by 20 weeks post-fertilization (22 weeks by LMP), the fetal brain has the full complement of neurons that are present in adulthood (Lagercrantz H et al. *Functional development of the brain in fetus and infant*. Lakartidningan 1991;88:1880-85).

At 19-20 weeks post-fertilization, electroencephalogram (EEG) recordings are possible (Flower MJ. *Neuromaturism of the human fetus*. J Med Philos 1985;10:237-251). We have no difficulty performing EEG studies on infants at this gestational age. At 22 weeks, continuous EEGs reflect awake and REM sleep state typical of neonate.

In the Neonatal Intensive Care Unit, we can witness first hand the change in vital signs associated with pain. When procedures such as IV placement or chest tube insertion are performed on neonates at 20 weeks post-fertilization age and above, the response is similar to those seen in older infants or children. With the advent of ultrasound including real-time ultrasound, we know that even at 8 weeks post fertilization, the fetus makes movements in response to stimuli. At 20 weeks post-fertilization, the fetus responds to sound, as mothers will commonly report increased fetal movement in response to music, sirens, or alarms.

At 23 weeks in utero, a fetus will respond to pain (intrahepatic needling, for example) with the same pain behaviors as older babies: screwing up the eyes, opening the mouth, clenching hands, withdrawal of limbs. In addition, stress hormones rise substantially with painful blood puncture, beginning at 18 weeks gestation (Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. "Fetal plasma cortisol and beta-endorphin response to intrauterine needling," *Lancet* 1994;344:77-81). This hormonal response is the same one mounted by born infants.

In addition, use of analgesia during neonatal surgery is standard of care; any infant undergoing fetal surgery is expected to receive appropriate pain medication as adults receive. In a 1992 study published in the *New England Journal of Medicine*, infants undergoing cardiac surgery had large increases in adrenaline, noradrenaline, and cortisol levels. Opioid analgesia markedly reduced these responses, as well as reduced peri-operative mortality.

Moreover, the fetus and neonate born prior to term may have an even heightened sensation of pain compared to an infant more advanced in gestation. There is ample evidence to show that while the pain system develops in the first half of pregnancy, the pain modulating pathways do not develop until the second half. It is later in pregnancy that the descending, inhibitory neural pathways mature,

which then allow for dampening of the pain experience. As reported in the *British Journal of Obstetrics and Gynecology*, the "... fetus may actually be more sensitive than the older child, and [this] may explain why the newborn shows exaggerated behavioral responses to sensory provocation" (*Br J Obs Gyn* 1999;106:881-886).

The idea that premature infants actually have greater pain sensitivity is supported by the fact that while pain transmitters in the spinal cord are abundant early on, pain inhibiting transmitters are sparse until later. (Anand KS, McGrath PJ, editors. *Pain Research and Clinical management*. Vol. 5. *Pain in neonates*. Amsterdam:Elsevier 1993:19-38). In addition, compared to the older infant, the premature infant requires greater concentrations of medications to maintain effective anesthesia. Thus, the fetus and premature infant appear to be even more susceptible to the pain experience.

In conclusion, I have no doubt that my premature neonatal patients feel and experience pain. Even early on, they demonstrate personalities and interact positively as well as negatively with their environments. With our advanced "views into the womb," we are now able to appreciate the active life of the developing fetus as one who is engaged with his or her uterine locale. I firmly believe, as the evidence shows, that the fetal pain experience is no less than the neonatal or adult pain experience. It may even be greater than that which you or I would experience from dismemberment or other physical injury.

One of the most basic of government principles is that the state should protect its members from harm. Technology, imaging, and clinical neonatology enable us to know much more about fetal life than ever before. We now understand the fetus to be a developing, moving, interacting member of the human family who feels pain as we do. If we are to be a benevolent society, we are bound to protect the fetus. We should not tolerate the gruesome and painful procedures being performed on the smallest of our nation.

Mr. FRANKS. Dr. Calhoun, you are recognized for 5 minutes, sir.

TESTIMONY OF BYRON C. CALHOUN, M.D., PROFESSOR AND VICE CHAIR, DEPARTMENT OF OBSTETRICS AND GYNECOLOGY, WEST VIRGINIA UNIVERSITY—CHARLESTON

Dr. CALHOUN. Chairman Franks and distinguished Members of the Subcommittee, I am Byron Calhoun. I serve as a professor and vice chair of obstetrics and gynecology at West Virginia University in Charleston. I am very pleased to have this opportunity to testify on the current issues, and am I very glad that I am able to speak for this consideration in the District of Columbia of the Pain-Capable Unborn Act.

I understand that this would limit abortion at 20 weeks fetal age, which is 22 weeks of LMP, which has already been discussed. Objections have been raised about this legislation saying that it should be permitted after 22 weeks because it is necessary and appropriate and a way to deal with a fetus with significant physical anomalies, including lethal anomalies, and I do not agree, emphatically. There are other ways that are far more humane for both the parents and the child.

My training, as noted, is in maternal-fetal medicine, which is the care exclusively of high-risk pregnancies, and this includes care of pregnancies, literally hundreds, with lethal anomalies. In my 25 years of practice, I have never found it necessary to terminate a pregnancy to save the life of a mother for anomaly. I have had to deliver multiple patients prematurely and had babies die from prematurity, but I have never had to take the life of a fetus to save the mother's life.

In the case of the fetal anomalies, we advocate patients be offered the option of perinatal hospice, which is the prenatal diagnosis for the terminally ill neonate in utero—excuse me, perinatal in utero, into perinatal hospice as a continuum of end-of-life care. Prior to the development of this concept, counseling provided parents with basically one option only, and that was assumed to be abortion, and offered no other alternatives. These were well-intentioned desires to spare the mother and her family, to solve the issue, to have the obstetrical provider do something, and perhaps deal with the discomfort they may have with bereaved parents, and perhaps the ill-advised avoidance of complications of pregnancy, and also an unsubstantiated concern of maternal mortality.

Research in grief actually has shown a different picture, and, in fact, there have been several studies show that there is actually prolonged and significant grief after the termination of a wanted pregnancy.

With regard to the fear of maternal mortality, the rates with induced abortion at the time we are talking are about 9 to 10 per 100,000, and the rates for pregnancy—for pregnancy death overall are about 10 per 100,000, and essentially the same mortality rate without an increase.

To do this we basically looked at Kubler-Ross' understanding of death and dying, and what we have done is support and give these patients an opportunity to be with their children in their pregnancy. We have used Saunders' idea that these people feared abandonment, and what we provide them is a high-touch care, not nec-

essarily high-tech. The emphasis is on affirming by care for these children and their families, and allowing them to have the support of medical, emotional, and spiritual needs of their family through a multidisciplinary team.

Its emphasis is in basically not a type of care, but basically in the amount of care, the focusing beyond the family, and not on the fetal diagnosis. The familyis placed at the center of the care and allowed to work through the grief and the death of their child.

Hospice preserves a time for bonding, and loving, and loss. Amy Kuebelbeck's writing of Waiting with Gabriel said with her son who had a fatal anomaly, "I know some people assume that continuing a pregnancy with a baby who will die is all for nothing, but it isn't all for nothing. Parents can wait with their baby. They can protect their baby and love their baby as long as that baby is able to live. They can give that baby a peaceful life and a peaceful goodbye. That is not nothing. That is a gift."

One of the major clinical issues in hospices I noted was fear. Patients really fear that they are going to be abandoned by their healthcare providers. They are also worried about pain, as was elegantly described by Dr. Malloy. With the ability to have perinatal hospice, we are able to develop birth plans, pain intervention, oxygen, feeding, medications, all the care that a normal neonate would have with the parents if they so desire through a multidisciplinary and easily accessible hospice team.

We also provide support for anticipatory grief, and we often shared the realistic outcomes of this pregnancy with the child with the lethal anomaly; usually diagnose—validate the diagnosis at delivery; and we allow these patients to spend the maximum amount of time with their children. We have published two series in this case with the children with lethal anomalies and found that if offered this implicitly, that between 70 and 85 percent of patients will choose a perinatal hospice.

In spite of what has been previously stated, there is a huge grassroots movement for this. There are now 125 perinatal hospices in 34 of the 50 States, and there are 13 international hospices. What had started as a small, simple idea, to promote patient-centered choice and humanity honoring care, has blossomed into a national and international movement for compassionate care for families. We look forward to the day when all patients will be allowed to be just patients and love their children for however long they may tarry.

Mr. FRANKS. Thank you, Dr. Calhoun.

[The prepared statement of Dr. Calhoun follows:]

Maternal – Fetal Medicine
Obstetrics and Gynecology
ROBERT C. BYRD
HEALTH SCIENCES CENTER
OF WEST VIRGINIA UNIVERSITY
Charleston Division

Testimony of Byron C. Calhoun, MD, FACOG, FACS, MBA
Professor and Vice Chair, Department of Obstetrics and Gynecology
West Virginia University-Charleston
Before the Subcommittee on the Constitution,
Committee on the Judiciary,
U.S. House of Representatives

May 17, 2012

Chairman Franks and distinguished members of the subcommittee, I am Byron C. Calhoun. I serve as a professor and as vice chair of the Department of Obstetrics and Gynecology at West Virginia University-Charleston. I am pleased to have this opportunity to testify on current issues that may arise during your consideration of the District of Columbia Pain-Capable Unborn Child Protection Act (H.R. 3803).

As you know, this legislation would prohibit abortion within the federal jurisdiction that it covers, beginning at 20 weeks fetal age, which is 22 weeks in the system of dating that is commonly employed in obstetrics, which counts pregnancy as beginning at the time of the last menstrual period (the "LMP" system). The bill contains an exception for certain cases in which an abortion is deemed necessary because of danger to the mother's life.

Objections have been raised to this legislation by some who say that abortion should be permitted even after 22 weeks LMP (20 weeks fetal age) because it is the necessary and appropriate way to deal with a fetus with significant physical anomalies including lethal anomalies. I do not agree. There are other alternatives that are far more humane for both parents and child.

My training, as noted in my biography, involves maternal-fetal medicine, which is the care of high risk pregnancies. This includes the care of pregnancies with lethal anomalies. In my almost 25 years of practice, I have never found it necessary to terminate a pregnancy to save the life of the mother for a fetal anomaly. I have had to deliver patients prematurely and had babies die from prematurity, but never had to take the life of a fetus to save the mother's life.

In the case of a fetal anomaly, we advocate patients be offered the option of the perinatal hospice, which is the prenatal diagnosis of a terminally ill fetus in-utero leading to perinatal hospice as part of the continuum of end-of-life care. Prior to the development of perinatal hospice, the counseling provided to parents facing such a diagnosis generally assumed abortion as the expected intervention, and offered no other alternative. There were the well-intentioned desires to "spare the mother and family" a distressing experience, a need to "get it over with," an obstetrical provider's need to "do something" and deal with the discomfort of bereaved patients, an ill-informed desire to avoid complications of pregnancy, and an unsubstantiated fear of increased maternal mortality.

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Research in grief after termination of pregnancy paints a much different landscape. Early, small studies provided an initial glimpse that termination losses were as intense as spontaneous losses. Zeanah, et al, 1993 reported a case-control study of 23 individuals and found a 17% (4/23) depression rate and 23% (5/23) seeking psychiatric counseling at two months.[1] A more recent study of 253 women from 2-7 years after termination of pregnancy for fetal anomalies prior to 24 weeks by Korenromp et al, 2005 found that pathologic grief persisted in 3% of patients (2/253) and that 17% (33/253) suffered from symptoms of posttraumatic stress.[2] Finally, Korenromp et al, 2009 found persistent and significant grief responses at 4,8, and 16 months.[3] At 4 months 46% of women revealed pathologic levels of posttraumatic stress symptoms and at 16 months 21% still had pathologic levels of posttraumatic stress symptoms.[3] In contrast, Janssen et al, 1996 published a study of 227 women with first trimester losses compared to a control group of 213 women matched for live births.[4] The first 6 months showed an increased level of depression, anxiety, and somatization in the miscarriage group, but by one year there was no difference between the 2 groups.[4]

With regard to the fear of increased maternal mortality, the mortality rates with induced abortion from 16-20 weeks are quoted as 9.3/100,000 live births and the rate for pregnancy related mortality is 10/100,000 live births.[5,6] So, essentially the mortality rates are the same for either of the management choices.

We utilized the seminal work of Kubler-Ross on modern medicine's understanding of death and dying to assist to shape our concept.[7] At the same time Kubler-Ross transformed the discussions around death, Saunders transformed the care of the dying with her modern hospice movement.[8] The unifying concept in hospice was the holistic approach to the physical, emotional, and spiritual support for dying patients and their families. The essence remained treating the dying with dignity and as if they really were alive and not yet dead. The patient and family's fear of abandonment could then be met. The philosophy of hospice has spread throughout the world. Its care may be found in various forms, institutions, and hospice in some manner may be found in almost every community today.

Perinatal hospice families who choose to carry their pregnancies in which the fetus has a lethal condition possess many of the same characteristics of families with a terminally ill adult or child, a clinical scenario in which hospice has been well accepted and a useful method of care. Many of the hospice principles were successfully applied in perinatal hospice. There was an emphasis on affirming life by care for the loved one while regarding dying as a normal process; a conscious effort to neither hasten death nor prolong dying; stressing values beyond the mere physical needs of the dying individual; allowing the parents to "parent" their child for whatever time they are allowed, and supporting the medical, emotional, and spiritual needs of the family through an organized multidisciplinary team that cares for the family after the death of the loved child during the period of grief.

The care in perinatal hospice differs in emphasis, not type of care from other modes of perinatal care. Its primary focus is on the family and not the fetal diagnosis. The family is placed in the center of the care and there is a continuum of support from the diagnosis, through death, and grief. It agrees with Knapp et al, that "dying involves real people, even unborn fetuses [and that] significant relationships are disrupted and familiar bonds are severed".[9] Hospice preserves time for the bonding, loving, and loss; time for parents to adjust to the dying process. Amy Kuebelbeck, author of *Waiting with Gabriel*,[10] a book about her own experience with her son who had a fatal form of hypoplastic left heart, notes, "I know that some people assume that continuing a pregnancy with a baby who will die is

all for nothing. But it isn't all for nothing. Parents can wait with their baby, protect their baby, and love their baby for as long as that baby is able to live. They can give that baby a peaceful life - and a peaceful goodbye. That's not nothing. That is a gift."[11]

One of the major clinical issues in hospice care remains fear. The patients who are dying fear abandonment, and in the same way, the perinatal hospice families fear abandonment and loss of relationships during the loss of their child. Hospice emphasizes they are allowed to "parent" their child how they would like to do so. We discuss the support of and care for them during their pregnancy, delivery, and death of their child. Parents also fear their baby might have pain. If they desire comfort measures for their baby: oxygen, feeding, medications, pain relief if indicated, and wound dressings; they are assured these will be provided. Some parents want to be seen when other patients are not present and some parents want to be with other pregnant women. Flexibility to the parents' wants and schedules is critical to the management of these pregnancies. Reduction of feelings of isolation and abandonment, through multidisciplinary and easy accessibility to the hospice team, are the mainstays of perinatal hospice care.

Instruction is given in anticipatory grief as well as ways to relate to other children in the family, friends, and family members. Often there remains a hope that the diagnosis is incorrect and that their child will be the miracle baby who somehow survives. Gentle sharing of the realistic outcome of the pregnancy is balanced with the hope for simplified dreams for their baby.

The grief accompanying a wanted child in the perinatal loss may be more intense than those with other losses. The lack of physical contact with, and minimal amount time with the fetus, may prevent connection within the family and minimize the feelings of loss. Memories built around the child are important in the grieving process. Frequent ultrasounds are provided of their baby, and, other family members are invited to attend; particularly grandparents and siblings, to come and see the baby. Seeing the baby cements the relationship and bond with the family and the child. Video tapes may be recorded for the family as the only living memories of their child.

Delivery plans are covered in detail with the parents. It is especially necessary for the parents to design their own birthing plan including a possible live birth. This may include fetal monitoring , which we usually do not recommend, unless the parents agree to possible cesarean delivery. Cesarean delivery may be offered in the event the parents want to see and hold their living child. If the parents are adequately counseled regarding the increased maternal risk for cesarean delivery, we will provide this service.

Diagnosis is validated at delivery and the family allowed to spend maximum time with their child. The time allows parents to contribute something special to their child's life and to let family members hold the infant and even perform its first (and maybe only) bath. The neonatal team may continue hospice care as well.

We have published two previous case series in perinatal hospice in diverse medical environments: a military medical center and a community based tertiary care medical center.[12,13] Our first series published in 2003 review our experience with a military population where we discussed 33 patients eligible for perinatal hospice care. Out of the 33 patients, 28 (85%) chose hospice care.[12] We had a 61% (17/28) live birth rate: 12 vaginal deliveries with 4 preterm (< 37 weeks) and 8 term; and 5 cesarean deliveries (18% or 5/28).[12] In our subsequent paper at a civilian tertiary care center we had 28 patients eligible for perinatal hospice with 75% (21/28) choosing hospice.[13] Out of our 21 patients

who chose hospice we had a 76% live birth rate (16/21) with 15 vaginal deliveries. Four of the deliveries were preterm (before 37 weeks) and 11 were full term. We had one cesarean section (1/21 or 5%) for maternal request of a live born baby. All our live born babies lived in the combined series (33 total live born) from 20 minutes to 256 days (one trisomy13). The majority of the neonates expired within 24 hours.[12,13] There were no maternal morbidities or mortalities in either of our series. This replicates previous authors' experience.[14]

The publication of our two case series provided the necessary clinical support for perinatal hospice demonstrating no increase in either maternal mortality or morbidity. A number of educational presentations have also been presented in various venues in support of the development of perinatal hospice. To date, 125 perinatal hospices in 34 of the United States and 13 international hospices have been created.[15] What started as a small, simple idea to promote patient-centered choice and humanity honoring care, has blossomed into a national and international movement for compassionate care for families.

We look forward to the day when all parents will be allowed to "just be parents" and love their children for however long they may tarry.

References

1 Zeanah CH, Dailey JV, Rosenblatt MJ, Saller DN. Do women grieve after terminating pregnancies because of fetal anomalies? A controlled investigation. Obstet Gynecol 1993;82(2):270-275.
2 Korenromp MJ, Page-Christiaens gCML, Van den Bout J, Mulder EJH, Visser GHA. Adjustment to termination of pregnancy for fetal anomaly: a longitudinal study in women in women at 4, 8, & 16 months. Am J Obstet Gynecol 2009;201:160.e1-7.
3 Korenromp MJ, Page-Christiaens gCML, Van den Bout J, Mulder EJH, Visser GHA. Adjustment to termination of pregnancy for fetal anomaly: a longitudinal study in women in women at 4, 8, & 16 months. Am J Obstet Gynecol 2009;201:160.e1-7.
4 H.J. Janssen et al., "Controlled Prospective Study on the Mental Health of Women Following Pregnancy Loss," *American Journal of Psychiatry* 153 (1996): 226–230.
5 Lawson HW, Frye A, Atrash HK, et al. (1994) Abortion mortality,United States, 1972 through 1987. Am J Obstet Gynecol 171:365-372.
6 CDC (1998). Maternal mortality--United States, 1982-1996. MMWR 47:705-707.
7 Kubler-Ross. On death and dying. New York: Macmillan Publishing Co; 1969.
8 Saunders C. The hospice: its meaning to patients and their physicians. Hosp Pract (Off Ed) 1981;16:93-108.
9 Knapp RJ, Peppers LG. Doctor-patient relationships in fetal/infant death encounters. J Med Edu 1979;54:775-80.
10 Kuebelbeck, A. Waiting with Gabriel: a story of cherishing a baby's brief life. Chicago, IL: Loyola Press; 2003.
11 Amy Kuebelbeck. Quote from meeting, "Perinatal Palliative Care with Compassion, Care & Confidence", 29-30 April 2009, Lancaster General Hospital, Lancater, PA.
12 Calhoun BC, Napolitano P, Terry M, Bussey C, Hoeldtke NJ. Perinatal hospice: comprehensive care for the family of the fetus with a lethal condition. J Repro Med 2003;48:343-348.
13 D'Almeida M, Hume RF, Jr., Lathrop A, Njoku A, Calhoun BC. Perinatal Hospice: Family-Centered Care of the Fetus with a Lethal Condition. J of Physicians and Surgeons 2006:11(3):52-55.
14 Spinatto JA, Cook VD, Cook CR, Voss DH. Aggressive intrapartum management of lethal fetal anomalies: beyond fetal beneficence. Obstet Gynecol 1995;85:89-92.
15 www.perinatalhospice.org (5/14/12)

Mr. FRANKS. And, Miss Zink, you are now recognize for 5 minutes.

TESTIMONY OF CHRISTINE (CHRISTY) ZINK, WASHINGTON, DC

Ms. ZINK. Good afternoon, Mr. Chairman, Representative Nadler, and other Members of the Committee. My name is Christy Zink.

I, like many women in the Washington, D.C., area, am a mother. Almost every day I rush around to get two kids woken up, dressed and out the door. Between my 5-year-old daughter and 11-month-old son, there are backpacks, diaper bags, milk bottles, juice boxes, lunch boxes, permission slips, and stuffed bunnies. There are also the mysterious hunt for two matching shoes and the eternal battle to actually get those shoes on two matching feet. I, like so many women, work diligently to balance family and work, and I feel lucky to have this challenge.

In addition to my two children, I was also pregnant in 2009. I would often wonder about whose eyes the baby might have, and who my child might grow up to be. I was looking forward to the ultrasound when we would get a chance to have a look at the baby in utero. I certainly hadn't anticipated that my husband and I would have to make the most difficult decision of our lives.

I took extra special care of myself during this pregnancy. I received excellent prenatal attention. Previous testing had shown a baby growing on target with the limbs and organs all in working order. However, when I was 21 weeks pregnant, an MRI revealed that our baby was missing the central connecting structure of the two parts of his brain. He specialist diagnosed the baby with agenesis of the corpus collosum.

What allows the brain to function as a whole was simply absent, but that wasn't all. Part of the baby's brain had failed to develop. Where the typical human brain presents a lovely rounded symmetry, our baby had small globular splotches. In effect, our baby was also missing one side of his brain.

I am fortunate to live in Washington, D.C., because my husband and I were able to consult some of the best radiologists, neurologists, and geneticists not just in our city or in the country, but in the world. We asked every question we could. The answers were far from easy to hear, but they were clear. There would be no miracle cure. His body had no capacity to repair this anomaly, and medical science could not solve this tragedy.

Our baby's condition could not have been detected earlier in my pregnancy. Only the brain scan could have found it. The prognosis was unbearable. No one could look at those MRI images and not know instantly that something was terribly wrong. If the baby survived the pregnancy, which was not certain, his condition would require surgeries to remove more of what little brain matter he had in order to diminish what would otherwise be a state of near-constant seizures.

I am here today to speak out against the so-called Pain-Capable Unborn Child Protection Act. Its very premise that it prevents pain is a lie. If this bill had been passed before my pregnancy, I would have had to carry it to term and give birth to a baby whom the doctors concurred had no chance of a life and would have experienced near-constant pain. If he had survived the pregnancy, which was not certain, he might never have left the hospital. My daughter's life, too, would have been irrevocably hurt by an almost always absent parent.

The decision I made to have abortion at almost 22 weeks was made out of love and to spare my son's pain and suffering. I am horrified to think that the doctors who compassionately but objec-

tively explained to us the prognosis and our options for medical treatment and the doctor who helped us terminate the pregnancy would be prosecuted as criminals under this law for providing basic medical care and expertise.

I live and work in Washington, D.C. My husband and I own a house here. We vote, and we believe in the democracy at the heart of this country. It is unconscionable that someone would come into my city from the outside and try to impose a law that doesn't represent the best interests of anyone, especially families like mine. This proposed law is downright cruel as it would inflict pain on the families, the women, and the babies it purports to protect.

It is in honor of my son that I am here today speaking on his behalf. And I am also fighting for women like me to have the right to access abortion care when we need to beyond 20 weeks, especially for those women who could never imagine they would have to make this choice. I urge you not to pass this harmful legislation.

Mr. FRANKS. Thank you, Ms. Zink.

[The prepared statement of Ms. Zink follows:]

Prepared Statement of Christine (Christy) Zink, Washington, DC

Good afternoon. Mr. Chairman, Representative Nadler, and other members of the committee. My name is Christy Zink. I, like many women in the Washington, DC area, am a mother. Almost every day, I rush around to get two kids woken up, dressed, and out the door. Between my five-year-old daughter and eleven-month-old son there are backpacks, diaper bags, milk bottles, juice boxes, lunch boxes, permission slips, and stuffed bunnies. There are also the mysterious hunt for two matching shoes and the eternal battle to actually get those shoes on two matching feet.

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I took extra special care of myself during this pregnancy. I received excellent prenatal attention. Previous testing had shown a baby growing on target, with the limbs and organs all in working order. However, when I was 21 weeks pregnant, an MRI revealed that our baby was missing the central connecting structure of the two parts of his brain. A specialist diagnosed the baby with agenesis of the corpus callosum. What allows the brain to function as a whole was simply absent. But that wasn't all. Part of the baby's brain had failed to develop. Where the typical human brain presents a lovely, rounded symmetry, our baby had small, globular splotches. In effect, our baby was also missing one side of his brain.

We are fortunate to live in Washington, DC, because we were able to consult some of the best radiologists, neurologists, and geneticists not just in our city or in the country, but in the world. We asked every question we could. The answers were far from easy to hear, but they were clear. There would be no miracle cure. His body had no capacity to repair this anomaly, and medical science could not solve this tragedy.

Our baby's condition could not have been detected earlier in my pregnancy. Only the brain scan could have found it. The prognosis was unbearable. No one could look at those MRI images and not know, instantly, that something was terribly wrong. If the baby survived the pregnancy, which was not certain, his condition would require surgeries to remove more of what little brain matter he had in order to diminish what would otherwise be a state of near-constant seizures.

I am here today to speak out against the so-called Pain-Capable Unborn Child Protection Act. It's very premise—that it prevents pain—is a lie. If this bill had been passed before my pregnancy, I would have had to carry to term and give

birth to a baby whom the doctors concurred had no chance of a life and would have experienced near-constant pain. If he had survived the pregnancy—which was not certain—he might have never left the hospital. My daughter's life, too, would have been irrevocably hurt by an almost always-absent parent.

The decision I made to have an abortion at almost 22 weeks was made out of love and to *spare* my son's pain and suffering.

I am horrified to think that the doctors who compassionately but objectively explained to us the prognosis and our options for medical treatment, and the doctor who helped us terminate the pregnancy, would be prosecuted as criminals under this law for providing basic medical care and expertise.

I live and work in Washington, DC. My husband and I own a house here, we vote, and we believe in the democracy at the heart of this country. It is unconscionable that someone would come into my city from the outside and try to impose a law that doesn't represent the best interests of anyone, especially families like mine. This proposed law is downright cruel, as it would inflict pain on the families, the women, and the babies it purports to protect.

It's in honor of my son that I'm here today, speaking on his behalf. I am also fighting for women like me, to have the right to access abortion care when we need to beyond 20 weeks—especially for those women who could never imagine they'd have to make this choice. I urge you not to pass this harmful legislation.

Mr. FRANKS. I now recognize myself for 5 minutes to begin questioning.

And, Dr. Levatino, I obviously was moved significantly by your testimony. And I think one of the great challenges that we have as human beings, we always seem to have as one or our greatest talents the ability to blind ourselves to a truth that we don't want to face. I know that is certainly true many times in my own life. And yet, in this place that should be something that we war against with all assiduous diligence, because the implications are pretty profound.

And one of the things that this bill does, and the discussion of it, seems to demonstrate the humanity of these little babies and the gross inhumanity of what is done to them. And I applaud your courage to come here as not only a former lawyer, but as someone that has performed abortions earlier. There is very few ways to try to impeach your sincerity or your credibility when you have gone 180 degrees here as you have done. And I appreciate what you have done.

So my first question is to you: The Criminal Code of the District of Columbia, section 22-1001, prohibits cruelty to animals, and with unanimous consent, I will enter a copy of this statute for the record.

[The information referred to follows:]

Submission for the hearing record, May 17, 2012, Subcommittee on the Constitution hearing on DC Pain-Capable Unborn Child Protection Act

DC ST § 22-1001
Formerly cited as DC ST 1981 § 22-801

➧§ 22-1001. Definition and penalty.

(a)(1) Whoever knowingly overdrives, overloads, drives when overloaded, overworks, tortures, torments, deprives of necessary sustenance, cruelly chains, cruelly beats or mutilates, any animal, or knowingly causes or procures any animal to be so overdriven, overloaded, driven when overloaded, overworked, tortured, tormented, deprived of necessary sustenance, cruelly chained, cruelly beaten, or mutilated, and whoever, having the charge or custody of any animal, either as owner or otherwise, knowingly inflicts unnecessary cruelty upon the same, or unnecessarily fails to provide the same with proper food, drink, air, light, space, veterinary care, shelter, or protection from the weather, shall for every such offense be punished by imprisonment in jail not exceeding 180 days, or by fine not exceeding $250, or by both.

(2) The court may order a person convicted of cruelty to animals:

(A) To obtain psychological counseling, psychiatric or psychological evaluation, or to participate in an animal cruelty prevention or education program, and may impose the costs of the program or counseling on the person convicted;

(B) To forfeit any rights in the animal or animals subjected to cruelty;

(C) To repay the reasonable costs incurred prior to judgment by any agency caring for the animal or animals subjected to cruelty; and

(D) Not to own or possess an animal for a specified period of time.

(3) The court may order a child adjudicated delinquent for cruelty to animals to undergo psychiatric or psychological evaluation, or to participate in appropriate treatment programs or counseling, and may impose the costs of the program or counseling on the person adjudicated delinquent.

(b) For the purposes of this section, "cruelly chains" means attaching an animal to a stationary object or a pulley by means of a chain, rope, tether, leash, cable, or similar restraint under circumstances that may endanger its health, safety, or well-being. Cruelly chains includes, but is not limited to, the use of a chain, rope, tether, leash, cable or similar restraint that:

(1) Exceeds 1/8 the body weight of the animal;

(2) Causes the animal to choke;

(3) Is too short for the animal to move around or for the animal to urinate or defecate in a separate area from the area where it must eat, drink, or lie down;

(4) Is situated where it can become entangled;

(5) Does not permit the animal access to food, water, shade, dry ground, or shelter; or

(6) Does not permit the animal to escape harm.

(c) For the purposes of this section, "serious bodily injury" means bodily injury that involves a substantial risk of death, unconsciousness, extreme physical pain, protracted and obvious disfigurement, mutilation, or protracted loss or impairment of the function of a bodily member or organ. Serious bodily injury includes, but is not limited to, broken bones, burns, internal injuries, severe malnutrition, severe lacerations or abrasions, and injuries resulting from untreated medical conditions.

(d) Except where the animal is an undomesticated and dangerous animal such as rats, bats, and snakes, and there is a reasonable apprehension of an imminent attack by such animal on that person or another, whoever commits any of the acts or omissions set forth in subsection (a) of this section with the intent to commit serious bodily injury or death to an animal, or whoever, under circumstances manifesting extreme indifference to animal life, commits any of the acts or omissions set forth in subsection (a) of this section which results in serious bodily injury or death to the animal, shall be guilty of a felony and, upon conviction thereof, shall be punished by imprisonment not exceeding 5 years, or by a fine not exceeding $25,000, or both.

➡§ 22-1013. Definitions.

In §§ 22-1001 to 22-1009, inclusive, and § 22-1011, the word "animals" or "animal" shall be held to include all living and sentient creatures (human beings excepted), and the words "owner," "persons," and "whoever" shall be held to include corporations and incorporated companies as well as individuals.

Mr. FRANKS. This statute explicitly covers, "all living and sentient creatures, human beings excepted," if a prosecutor can prove, "serious bodily injury," or if a prosecutor can prove, "to an animal or indifference to animal life;" that a single offense can be punished by up to 5 years in prison or a fine not to exceed $25,000 or both. Serious bodily injury includes, among other things, the infliction of, "extreme physical pain or mutilation, or broken bones, or severe lacerations."

Now, I heard your vivid description of the D&E abortion method, which I am told is the most frequent method used for abortion after 20 weeks, and it seems clear that it follows this description of mu-

tilating and breaking bones, lacerating, and worse, and we have heard very convincing evidence that it would inflict, quote, "extreme physical pain."

Now, that fits all of the criteria, and I find it a tremendous—I don't even want to use the word "irony"—just a break from human compassion that while we would do the right thing and prevent those things from happening to children—to animals but not to human babies. And I am just wondering if you think that my equating the two has any parallel, and how you would respond to it yourself.

Dr. LEVATINO. Not at all, Mr. Chairman.

The abortion debate is obviously a very uncomfortable topic for many. It is a very hot political topic. There are very strong feelings on both sides.

I have been on both sides of this issue. I do understand both sides. It is a tremendous irony—the word seems inadequate—that, as you say, feed animals get more—you know, get more consideration than unborn humans.

Even as an abortionist, when I learned to do D&E abortions, I have to tell you, the only word I can express, even as an experienced physician for many years at that point, was in doing a D&E abortion, it is absolutely gut-wrenching for the physician. It is easier on the patient for sure, and that was one of the advantages of the procedure.

We wanted a procedure like D&C where a patient would basically go to sleep, wake up, and it would all be over. And it certainly was better from the standpoint of the patient, from that standpoint, is one of the strengths of the procedures, one of the reasons we do them. But to literally tear a human being apart with your own hands—I would invite the Committee to handle this instrument. This is the identical instrument I used. It is an absolutely gut-wrenching procedure. And I agree with you, it is, to me, unconscionable to say we give more consideration to feed animals than we do to human beings.

Mr. FRANKS. Well, Dr. Levatino, you know, in responding to your earlier comment that this is unconstitutional, the courts have stated that States have an interest in forbidding medical procedures in which the State's reasonable determination might cause the medical profession or society as a whole to become insensitive, or even disdainful, to life, including life in the human fetus. A State may take measures to ensure the medical profession and its members are viewed as healers, sustained by compassionate and rigorous ethics, and cognizant of the dignity and value of each human life, even life which cannot survive without the assistance of others. That seems to describe what we are trying to accomplish here.

Do you think, in your mind, that doing late-term abortions can create the impression that causing the medical—or create the trend in the medical profession or society as a whole to become insensitive and even disdainful of life, including life in the human fetus? What is your perspective?

Dr. LEVATINO. I would completely agree with that. As a physician, I used to teach students and I used to tell them, you know, you have learned to maintain a certain distance between you and your patients. I think that you start learning it on day one in anat-

omy class, where you are literally taking apart a human body, and you don't think of it as, you know, this was—you see it as a collection of organs, and you don't see this as somebody's son, or daughter, or husband or wife.

It was the same way. As I said, the procedures are very gut-wrenching, but I guess you can get used to anything over time.

I do agree that there is a great insensitivity toward life. It has become an engrained part of our culture, and this simply adds to that.

Mr. FRANKS. Thank you, Doctor, and I will now yield to the Ranking Member for 5 minutes for questions.

Mr. NADLER. Thank you.

Ms. Zink, first of all, I want to thank you for agreeing to testify today. As a parent, your story was very difficult to listen to, and I can't even begin to imagine how difficult it must have been to live through it, much less come here and describe your experience to some very unsympathetic people. So I want to thank you for your willingness to put a human face on this question, and for your courage in being here.

One of the really harmful consequences of this bill is that there are some fetal conditions that cannot be diagnosed before the 20th week of pregnancy. In those situations the tragedy of learning that there is, for example, a fetal anomaly that is incompatible with life is compounded by the fact that this bill would make it impossible to receive abortion care if that is the medically indicated treatment. In fact, isn't it correct that the diagnosis in your case could not have been made before the 20th week?

Ms. ZINK. That is correct.

Mr. NADLER. If this bill had been law when you had to face your ordeal, your doctor would have had to risk jail and a lawsuit to provide you with the medical services that you required. Would you care to comment on that?

Ms. ZINK. If I pause it is because it is so horrible that the idea that you cannot have a conversation with your doctor who knows you, who knows your medical history, who can look at the medicine, and who can speak from his expertise; that all of a sudden the things that we take for granted about working with your doctor, about going to someone who has that trained expertise, about having a relationship with your doctor, that all of that suddenly becomes criminal, to me, is just beyond belief.

Mr. NADLER. Thank you.

I would like to ask a couple of questions of all of the doctors, one at a time.

Dr. Levatino, yes or no, do you believe that your views with respect to when fetuses feel pain are now established and generally accepted by the scientific community, or is yours the minority view?

Dr. LEVATINO. As far as I am concerned, Congressman, they are accepted by the scientific community——

Mr. NADLER. Thank you.

Dr. Malloy?

Dr. LEVATINO [continuing]. And based on experience as well.

Mr. NADLER. Dr. Malloy?

Dr. MALLOY. I can guarantee you that any baby who is receiving some procedure in a NICU——

Mr. NADLER. That is not what I asked. We heard your view. Do you believe that your views are now established and generally accepted, or are you a minority view?

Dr. MALLOY. Which view would that be?

Mr. NADLER. As to when pain is felt.

Dr. MALLOY. That a preemie feels pain?

Mr. NADLER. Not a preemie. A preemie at 20 weeks. A preemie at 20 weeks in utero, excuse me. A fetus at 20 weeks in utero that feels pain. You stated your opinion on that. Do you think that your opinion now is generally accepted by the scientific community, or do you think that your view is a minority view?

Dr. MALLOY. I spoke about the pain that the fetus and the premature infant feels, so I am not separating those two things. So I think my view is the majority view, that——

Mr. NADLER. Okay.

And Dr. Calhoun.

Dr. CALHOUN. I believe mine is also the majority view.

Mr. NADLER. Thank you.

Then, all three of you, how do you explain—I shouldn't say that. Are you aware of the research published in the Journal of the American Association and the conclusions of the Royal Academy of Obstetricians and Gynecologists among others? I am not asking if you agree or disagree. Are you aware of it?

Dr. Levatino?

Dr. LEVATINO. I am well aware of the paper that was published in 19-—or, excuse me, 2005, by—in JAMA, sir. There were serious problems with that paper, not the least of which——

Mr. NADLER. I just asked if you are aware.

Dr. Malloy, are you aware of it?

Dr. MALLOY. I am sorry?

Mr. NADLER. Are you aware of the research published by Journal of the American Medical Association and the conclusions of the Royal Academy of Obstetricians and Gynecologists?

Dr. MALLOY. Yes. I read the paper in JAMA.

Mr. NADLER. Thank you.

Dr. Calhoun.

Dr. CALHOUN. I have read the paper in JAMA as well.

Mr. NADLER. Okay. Now, since the paper in JAMA, the Journal of the American Medical Association, says that evidence regarding the capacity for fetal pain is limited, but indicates that fetal perception of pain is unlikely before the third trimester, and the conclusion of the Royal Academy of Obstetricians and Gynecologists concluded, quote, "It can be concluded that the fetus cannot experience pain in any sense prior to 24 weeks gestation," then you are saying that those are minority views, and they are clearly wrong.

Dr. LEVATINO. I am saying that that is one paper, Congressman, out of many.

Mr. NADLER. Dr. Malloy?

Dr. LEVATINO. And that paper has serious flaws, including the fact that the chief author was a medical student, who happened to previously be a lawyer for a prochoice——

Mr. NADLER. I only have 5 minutes, and I asked you simple questions. I don't need lectures.

Dr. Malloy, so your opinion is contrary to that expressed by the American Medical Association and the Royal Academy of Obstetricians and Gynecologists. Do you regard their view or yours as the minority view?

Dr. MALLOY. I believe there are serious flaws with that paper.

Mr. NADLER. Okay. But is theirs the majority or minority view in the field?

Dr. MALLOY. In my field of neonatology, mine would be the majority, and theirs would be the minority.

Mr. NADLER. Thank you. It would be the majority view in your field is what you just said?

Dr. MALLOY. Mine would be the majority view, not theirs.

Mr. NADLER. Yours would be the majority view.

Dr. Calhoun.

Dr. CALHOUN. Mine would be the majority view, not JAMA. That is a single paper.

Mr. NADLER. Okay, my last question.

Mr. CHABOT. Point of order. Hasn't the gentleman's time expired?

Mr. FRANKS. You are correct, and we may be able to have time for an additional round of questions, but I now recognize the gentleman from Ohio for 5 minutes for his questions.

Mr. CHABOT. Thank you very much.

And the gentleman from New York was talking about treating people rudely before. Let me try to be polite to the gentleman, the doctor here, and allow him to answer the question that was posed. I think you were saying something about the JAMA study, and what was that that you were going to say, Doctor?

Dr. LEVATINO. I am afraid that medical research isn't as free of politics as we wish it was. This is one paper. There are other papers that say quite the opposite. I thought that that paper was very interesting, and that the chief author was a medical student who was formerly an attorney who worked for NARAL. One of authors, the other authors, of that paper, a Dr. Drey, is one of the largest abortion providers in the city of San Francisco. I would hardly find their findings unbiased.

Mr. CHABOT. Thank you.

Let me ask further. Ms. Zink was relating her story, which was certainly moving, I think, to everybody in this room. She was talking about an unborn child that had, I would assume, a particularly rare condition. Would that be—would one of the doctors here like to at least tell us, is this something that is common in this particular case, or something that is relatively rare?

Dr. Calhoun.

Dr. CALHOUN. The agenesis of the corpus collosum?

Mr. CHABOT. Yes.

Dr. CALHOUN. It is relatively rare, but it is not that rare. I see it not infrequently in my care.

Mr. CHABOT. One out of what are we talking here?

Dr. CALHOUN. I would have to go back and look at it. I mean, I would have to go back and look. Maybe a half a percent or so.

Mr. CHABOT. We talking about 1 out of 200, if you mean ½ percent. Okay.

Dr. CALHOUN. Yes, sir.

Mr. CHABOT. Let us talk about the other 199, and maybe not all 199. And let me go back to you, Dr. Levatino, if I can. You mentioned, I think, 1,200 abortions that you had performed?

Dr. LEVATINO. Yes, sir.

Mr. CHABOT. And I don't want to put you on the spot here, but most of those abortions, is it safe to say that had they not been terminated through an abortion, that these would have been normal, healthy babies ultimately in the majority of those cases? Is that accurate, would you say?

Dr. LEVATINO. Yes, sir. That is typical with an abortion practice. It is certainly was with mine. The number of abortions out of the 1,200 that I did for fetal anomalies were less than 5.

Mr. CHABOT. Less than five. So we are talking about 1 out of 200 here. We are talking about the—out of the 1,200, what would you say would have typically been healthy babies?

Dr. LEVATINO. The vast majority. Over 99 percent, sir.

Mr. CHABOT. Okay. And so if we are looking at tragedies here, I mean, I think we have to look at the relative tragic situation that we are talking about. And, again, I don't want to put you on the spot, Doctor, but would you want to share—and if you don't want to, you don't have to—was there something in particular that changed your view on this important topic?

Dr. LEVATINO. I won't elaborate considerably. All I can say is, Ms. Zink, I do understand your pain. I have lost a child, too. I know what that feels like, and I am sorry.

It was a time, as I said, that I was very prochoice. This was a decision between a doctor and a patient, and nobody, including the baby's father, had anything to say about it. I was very dedicated in that business, and I did it for many years.

Going through this, doing that procedure, didn't exactly help me sleep at night. And in 1986, I lost a daughter. And after you have lost a child, and then you go back to the hospital—it was maybe 2 weeks after her death when I went back to work, and I went into the medical center to do my first D&E abortion.

And I reached in with that Sopher clamp, and I literally ripped out an arm or a leg. I got sick.

You know, when you do an abortion, you can't stop. You have to finish that abortion. If you don't, if you don't get all the pieces, your patient is going to come back infected, bleeding or worse.

And I know it sounds strange to people, but I tell you it is sincere, true and firsthand. For the first time in my career, after 1,200 abortions in private practice much less the hundreds I did during my training, I really looked at the pool of goo at the side of table that used to be somebody's son or daughter, and that was a very life-changing experience.

Mr. CHABOT. Thank you, Doctor.

And, Dr. Malloy, finally, before I run out of time, would you describe again as far as the pain what you said—you see this every day. What kind of pain are we talking about? How do you know there is pain there?

Dr. MALLOY. Well, we have to put IVs in babies, we put chest tubes in babies, we intubate babies, we do lots of things that are nowhere near dismemberment or stabbing them in the heart with

potassium chloride. We do things that are probably 100 as painful as what he is describing. And they feel that, they wince, they cry, they move away from it, they try to push your hand away when you are putting an IV in. So I know they respond to those simple procedures that we perform, so I can just shudder to think what is happening when that kind of procedure is performed.

Mr. CHABOT. Thank you, Doctor.

Mr. FRANKS. Thank you, Mr. Chabot.

You know, as I heard fetal anomaly being one of the prime justifications for all this, as someone that owes the medical community a great deal in life because of being born with a significant fetal anomaly myself, I have to tell you sometimes when I hear testimony like Dr. Levatino's, I sense two things: one, a sense of hope; and, two, difficulty in understanding how we got where we are.

With that, I would recognize Mr. Scott of Virginia for 5 minutes.

Mr. SCOTT. Thank you, Mr. Chairman.

Chairman, I notice that all of the—that none of the panelists are attorneys, and I was wondering if anybody on the panel is qualified to discuss the constitutionality of the legislation and how it would conform or not conform to U.S. Supreme Court cases. Okay?

Second question, is there anything unique about Washington, D.C., that this proposal should apply to Washington, D.C., and nowhere else?

Dr. LEVATINO. It wouldn't be true to say no or else that this legislation applies to D.C., but these similar legislations have been passed in other States.

Mr. SCOTT. Well——

Dr. LEVATINO. This is not the first time that I am aware of.

Mr. SCOTT. We are considering legislation justifying it to Washington, D.C., rather than the entire Nation. Is there anything unique about Washington, D.C., where we ought to have this proposal apply to D.C. and nowhere else?

Let me ask another question. This applies to abortions—as I understand the legislation, abortions performed in Washington, D.C. Would the prohibition apply for a Virginia resident coming into Washington, D.C., to get an abortion?

Dr. LEVATINO. As far as I know, yes, but I don't know for sure.

Mr. SCOTT. Okay. Would it apply to a Washington, D.C., resident going to Virginia to get an abortion?

Dr. LEVATINO. No, it would not.

Mr. SCOTT. It would not, okay.

Would it apply if the pregnancy resulted from rape?

Dr. LEVATINO. Yes.

Mr. SCOTT. Would it apply if the pregnancy resulted from incest?

Dr. LEVATINO. Yes.

Mr. SCOTT. And it would also apply, as I understand it, to a fetal medical condition inconsistent with life?

Dr. CALHOUN. Yes.

Mr. SCOTT. It would?

Dr. CALHOUN. Yes.

Mr. SCOTT. Thank you, Mr. Chairman.

I have no further questions, and I yield back.

Mr. FRANKS. Thank you, Mr. Scott.

And I would now recognize Mr. King for 5 minutes.

Mr. KING. Thank you, Mr. Chairman, and I thank the witnesses.

And I would like to go to Dr. Levatino, who has provided some very moving testimony here today, and ask that the procedures that you conducted over those years, 1,200-plus by your testimony, do you know of material that has been gathered, such as video of—for the procedures that you described here today?

It just occurred to me as I am listening to your testimony, of all the discussions that we have had, I don't recall ever a video being offered that might more vividly describe what you so vividly described.

Dr. LEVATINO. Am I aware of the existence of such material?

Mr. KING. Yes.

Dr. LEVATINO. It may well be out there, but I couldn't quote any for you.

Mr. KING. And isn't it common for medical procedures to be available on YouTube or other medical—let us see, I looked up here medical videos. There is at least one Web site that delivers a whole number of different medical procedures. You are not aware that anything is available on the open Web?

Dr. LEVATINO. Such things are generally available, but I haven't researched them to tell you where they are.

Mr. KING. I would ask if anybody on the panel is aware of any videos of this procedure on the open Web?

Dr. CALHOUN. None that I am aware of.

Mr. KING. Dr. Malloy? No?

Do you suspect that there is a concerted effort it to make sure that that information is not available, Dr. Levatino?

Dr. LEVATINO. I would be speculating. Let me put it this way: I think that when people see things—you can hear a description, but when you see things, when you actually see it, it tends to have a much greater impact.

I mean, the one thing I can think of that just happened to pop in my head is child labor laws. I mean, it is photographs that so many decades ago got us to change the child labor laws. I think the same thing can happen with any area of life, and especially this one. I often tell people I swear some people think the doctor waves his hand and the baby disappears. It just doesn't happen that way.

Mr. KING. One more question with Dr. Levatino, and if it is too personal I—decline to respond if you prefer, but how old was your daughter when you lost her?

Dr. LEVATINO. Just sort of her sixth birthday.

Mr. KING. Thank you very much, Doctor. I think I am going to close my questioning with that. It has been a very powerful testimony here today, and I yield back.

Mr. SCOTT. Mr. Chairman?

Mr. FRANKS. Mr. Scott.

Mr. SCOTT. Mr. Chairman, I would ask unanimous consent to enter into the record a letter and accompanying documents on behalf of the gentleman from Illinois, who was here earlier and had to leave. One is from Catholics for Choice.

Mr. FRANKS. Without objection.

Mr. SCOTT. Thank you.

[The information referred to follows:]

CATHOLICS
FOR
CHOICE

IN GOOD CONSCIENCE

May 17, 2012

US House of Representatives Committee on Judiciary
Subcommittee on the Constitution
H2-362 Ford House Office Building
Washington, DC 20515

Dear Chairman Franks, Ranking Member Nadler and Members of the Subcommittee:

On behalf of Catholics for Choice, I strongly urge you to oppose HR 3803, the misleadingly-titled "District of Columbia Pain-Capable Unborn Child Protection Act."

As Catholics, we believe that it is critical to stand with all women, Catholic and non-Catholic alike, who need later abortion care. The social justice tradition, deference to religious freedom and respect for each individual's conscience that are central to our faith compel us to do so.

Creating arbitrary gestational limits on when women can receive abortion care, as HR 3803's proposed restrictions would do, will unfairly target the District of Columbia's most vulnerable women, who may not have the financial resources to seek services elsewhere. By refusing even to provide exceptions in cases of rape, incest, fetal abnormalities or mental illness, this bill also assumes a draconian posture toward those very women whose circumstances most necessitate compassion, the ability to avail themselves of all medical options and respect for their conscience-based decisions.

Women need later abortions for many reasons, and these reasons will not diminish despite legislative attempts to arbitrarily restrict access to safe medical care. Women seeking later abortions may find themselves in any number of particularly difficult circumstances—when a doctor's visit for a wanted pregnancy reveals serious complications; when lack of insurance or Medicaid coverage necessitate that a woman with limited economic means must delay while saving the money to pay for her procedure; when a young woman, afraid of the consequences of revealing her pregnancy, has finally spoken up and sought medical care. Any woman who finds herself in need of a later abortion should be able to receive the care she needs. HR 3083 would deny that care, infringe upon the rights of the women of the District of Columbia and blatantly disrespect the conscience of any woman who decides to seek abortion care as well as any medical professional who wishes to provide it.

The majority of the more than 580,000 Catholics who live in the DC metropolitan area and the more than 68 million Catholics in the United States support policies that enable women and men to make their own decisions about whether and when to have children.

They oppose measures such as HR 3803 that would infringe upon the ability of each individual to follow his or her own conscience.

1436 U Street NW, Suite 301 • Washington, DC 20009 • tel 202-986-6093 • fax 202-332-7995
cfc@catholicsforchoice.org • catholicsforchoice.org

You have an opportunity to do the right thing by the majority of Catholic voters, who want their elected officials to listen to them, not the bishops, when making public policy, especially concerning women's health. You also have an opportunity to do the right thing for the women of the District of Columbia, whose elected official in Congress has already listened to her constituents and heard that HR 3803 is not what they want. I hope that you will do the same.

Enclosed are two articles from *Conscience* magazine that I hope will shed further light on this issue: "A Perspective on Later Abortion … From Someone Who Does Them," by Dr. Willie Parker, an obstetrician-gynecologist who serves women in the DC area; and "Fetal Pain?" by Dr. Stuart Derbyshire, a psychologist and expert in these issues. If you would like more information or have any questions, please do not hesitate to contact our domestic program director, Sara Hutchinson, at 202-986-6093 or by e-mail at SHutchinson@catholicsforchoice.org.

Sincerely,

Jon O'Brien
President

Enclosures: Derbyshire, Stuart. "Fetal Pain?" *Conscience*, XXXI No. 3, 2010.

Parker, Willie. "A Perspective on Later Abortion … From Someone Who Does Them." *Conscience* XXXIII No. 1, 2012.

Mr. NADLER. Mr. Chairman, I ask unanimous consent to insert into the record a report by the Royal College of Obstetricians and Gynecologists concluding that the cortical connections are not established; therefore, pain cannot be felt at this stage.

Mr. FRANKS. All right. Thank you.

[The information referred to follows:]

Royal College of Obstetricians and Gynaecologists

Fetal Awareness

Review of Research and Recommendations for Practice

March 2010

Fetal Awareness

Review of Research and Recommendations for Practice

REPORT OF A WORKING PARTY

March 2010

Royal College of Obstetricians
and Gynaecologists

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Glossary

4-D (four-dimensional) images	Three-dimensional images that move in real time (time being the fourth dimension)
anencephalic fetus	A fetus with the major part of the brain missing
anoxic stress	Physiological stress through lack of sufficient oxygen
anterior cingulate	A higher cortical (brain) structure responsible for processing the unpleasantness of pain
arborisation	Branching – in this case of nerve fibres growing into a brain region; this is required before all the correct connections can be formed
auditory cortex	The part of the brain responsible for processing sound
axons	'cables' or nerve fibres connecting different parts of the brain
brainstem	A lower brain structure, lying between the spinal cord and the thalamus which is responsible for many reflex actions such as breathing
catecholamines	A chemical typically released during stress
cerebral cortex	A sheet of densely packed neuronal cells which form the outer, folded part of the brain associated with higher functions
cognition/cognitive	Thinking, knowing, sensing and perceiving
cortical plate	Develops before the cerebral cortex proper
EEG (electroencephalogram)	Measures electrical discharges in the brain. Electrodes are placed on the scalp of a subject and the activity of the neurons in the underlying cortex is recorded
electrophysiological	Techniques used to directly record the electrical activity of the peripheral or central nervous system in the body
endocrine	Hormone circulating in the body
endorphins	A neurochemical released naturally in the body that, in adults, suppresses pain
endoscopic laser ablation	A technique for destroying tissues directed by a small telescope inserted into the body
ex utero intrapartum treatment	Delivery of the head and shoulders at caesarean section so that surgery can be performed while the baby is still receiving oxygen from the placenta
fetal magnetoencephalography	A technique to measure brain activity in fetus
haemodynamic	The movement of blood
hypoxaemia	Decreased blood oxygen

hysterotomy	Surgical incision in the uterus, usually to remove the fetus
insular cortex	Part of the cerebral cortex believed to be responsible for integrating sensory information
fMRI (functional magnetic resonance imaging)	A technique for measuring blood flow in the brain, which is indirectly related to neuronal activity
neurobiological	A generic term relating to the biological functions of the central nervous system
neuronal connection	A communicative contact between two neurons
neuropsychological	A psychological function associated with a part of the brain
nociceptor activity	Passage of electrical signals through a nerve fibre that detects noxious stimuli
noxious stimuli	Stimuli that do or could cause damage to the body
opiate/opioid	A neurochemical that suppresses pain, of which endorphins are an example
sensory cortex	Part of the cortex responsible for processing sensory stimuli from the body, such as touch
sentience	The ability to detect and experience a sensory stimulus
somatosensory	The senses that are detected on the surface or deep within the body, such as touch, temperature, pressure
spinothalamic pathways	Major pathway transmitting noxious information through the spinal cord
stress/stress response	Typically the release of catecholamines following an adverse event but may also include other chemical and behavioural responses
subcortical sensory nucleus	A part of the brain between the spinal cord and cortex that processes sensory information, such as the thalamus
subplate zone	A developmental structure that holds and guides neurons to their correct place in the cortex
synapse	A communication juncture between two neurons
thalamic	Pertaining to the thalamus
thalamus afferents	Fibres carrying information into the thalamus
transient tachypnoea	Rapid breathing observed shortly after birth indicating a temporary difficulty with respiration
venepuncture	Penetrating a vein for injection or for withdrawal of blood
viability	Ability to survive
visual cortex	Part of the cortex responsible for processing vision

Attention is also drawn to the glossary entitled *Medical Terms Explained* available on the RCOG website: www.rcog.org.uk/womens-health/patient-information/medical-terms-explained.

Summary

The need to review the 1997 RCOG Working Party Report on Fetal Awareness arose following discussion during the House of Commons Science and Technology Committee Report on Scientific Developments relating to the Abortion Act 1967. In accepting the findings and conclusions of the House of Commons report, the Minister of State for Public Health recommended that 'the College review their 1997 report into fetal pain'. Accordingly, this Working Party was established with the remit and membership described. The intention was to review the relevant science and clinical practice relevant to the issue of fetal awareness and, in particular, evidence published since 1997. In so doing, the report was completely rewritten, not only to take account of recent literature but also the evidence presented to the House of Commons Committee.

In reviewing the neuroanatomical and physiological evidence in the fetus, it was apparent that connections from the periphery to the cortex are not intact before 24 weeks of gestation and, as most neuroscientists believe that the cortex is necessary for pain perception, it can be concluded that the fetus cannot experience pain in any sense prior to this gestation. After 24 weeks there is continuing development and elaboration of intracortical networks such that noxious stimuli in newborn preterm infants produce cortical responses. Such connections to the cortex are necessary for pain experience but not sufficient, as experience of external stimuli requires consciousness. Furthermore, there is increasing evidence that the fetus never experiences a state of true wakefulness *in utero* and is kept, by the presence of its chemical environment, in a continuous sleep-like unconsciousness or sedation. This state can suppress higher cortical activation in the presence of intrusive external stimuli. This observation highlights the important differences between fetal and neonatal life and the difficulties of extrapolating from observations made in newborn preterm infants to the fetus.

The implications of these scientific observations for clinical practice are such that the need for analgesia prior to intrauterine intervention, for diagnostic or therapeutic reasons, becomes much less compelling. Indeed, in the light of current evidence, the Working Party concluded that the use of analgesia provided no clear benefit to the fetus. Furthermore, because of possible risks and difficulties in administration, fetal analgesia should not be employed where the only consideration is concern about fetal awareness or pain. Similarly, there appeared to be no clear benefit in considering the need for fetal analgesia prior to termination of pregnancy, even after 24 weeks, in cases of fetal abnormality. However, this did not obviate the need to consider feticide in these circumstances and, in this respect, further recommendations of relevance are included in the parallel report on *Termination of Pregnancy for Fetal Abnormality*.

Background

Remit

The Working Party was established in May 2008 with the following remit:

1. To review the RCOG Working Party Report *Fetal Awareness*, published in October 1997.
2. To review all evidence submitted to the Science and Technology Committee relating to the Abortion Act 1967.
3. To review all other evidence of relevance to fetal awareness and pain.
4. To publish a report based on the Working Party's findings.

The Working Party met on four occasions between July 2008 and July 2009 and reported to Council in November.

Membership

The Membership of the Working Party was:

Professor Allan Templeton FRCOG (Chair)

Professor Richard Anderson FRCOG, Reproductive Medicine Specialist, University of Edinburgh

Ms Toni Belfield, Member of the RCOG Consumers' Forum

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Professor Neil Marlow, Neonatologist, UCL London

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Ms Stephanie Michaelides, Royal College of Midwives

Supported by Mrs Charnjit Dhillon, RCOG Director of Standards, and Miss Maria Finnerty, Secretary to the Working Party

This report was peer reviewed by the following individuals, to whom the Working Group wishes to express gratitude:

Professor David Archard, Professor of Philosophy and Public Policy, Lancaster University

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1. Introduction

Following concerns generated by the debate on fetal awareness and, particularly, the controversy around whether the fetus could feel pain, the RCOG published, in October 1997, a working party report.[1] A guiding principle in that report was concern that the fetus should be protected from any potentially harmful or painful procedure but, at the same time, the assessment of the capacity to be harmed should be based on established scientific evidence. A major and important conclusion of the report was that the human fetus did not have the necessary structural integration of the nervous system to experience awareness or pain before 26 weeks of gestation. In addition, the report recommended that those carrying out diagnostic or therapeutic procedures on the fetus *in utero* at or after 24 weeks should consider the need for fetal analgesia.

This guidance was welcomed by the clinical and scientific communities, although, in recent years, the report has from time to time come under criticism in some quarters for being out of date and perhaps not having assessed all the known scientific evidence. This criticism has been most evident in discussing the age of viability (at present taken as 24 weeks of gestation in the UK) and the upper gestational limit in the context of induced abortion. The House of Commons Science and Technology Committee, in its report on *Scientific Developments Relating to the Abortion Act 1967* (published in October 2007),[2] made a number of important conclusions and recommendations, including some of direct relevance to this issue: 'We conclude that, while the evidence suggests that foetuses have physiological reactions to noxious stimuli, it does not indicate that pain is consciously felt, especially not below the current upper gestational limit of abortion. We further conclude that these factors may be relevant to clinical practice but do not appear to be relevant to the question of abortion'.[2]

A minority report, however, recorded in the minutes of the Committee on 29 October 2007 said, 'We are deeply concerned that the RCOG failed to give full information to the House of Commons Select Committee…since 1997 the RCOG has consistently denied that foetuses can feel pain earlier than 26 weeks, without acknowledging that amongst experts in this field there is no consensus. Professor Anand is a world authority in the management of neonatal pain and has put forward a cogent argument suggesting that the RCOG position is based on a number of false or uncertain presuppositions'.[1]

In the Government response to the House of Commons report (released November 2007) the Minister of State for Health welcomed the report and its conclusions and recommendations but importantly also indicated that 'we note the Committee's findings and are in agreement that the consensus of scientific evidence with regard to fetal pain at gestations below 26 weeks and we will be commissioning the College to review their 1997 working party report into fetal pain which will re-examine the latest evidence, much of which has been considered by the Committee, and any new research currently underway'.[3]

Accordingly, a Working Party was formed to review the 1997 report. At its first meeting it decided to review not only the evidence in the original report but also, more importantly, any relevant evidence published since, including particularly the literature referred to in the minority report. As with the original report, it was decided not to reconsider the ethical situation

surrounding viability and abortion, not least because many of the relevant issues had been addressed in the Nuffield Council publication *Critical Care Decisions in Fetal and Neonatal Medicine: Ethical Issues* (2006).[4] Their terms of reference centred on the ethical, social, economic and legal issues arising from recent developments in fetal and neonatal medicine relating to prolonging life, as well as issues raised by advances in research and practice. This discussion very much revolved around 24 weeks as the age at which survival without impairment becomes more likely and, with the acceptance that survival without serious impairment or disability is highly unusual at 22 weeks of gestation, this led to the conclusion that there was no obligation to attempt resuscitation at gestational age of 23 weeks or lower. Importantly, the report recommended that a group of specialists and interested parties should develop a definition of 'born alive', with consideration to incorporating such a definition in statute. The RCOG has now considered this issue and intends to pursue further discussion with the Department of Health in relation to the clinical and legal consequences.

Furthermore, the Working Party agreed that, in reviewing past and current evidence, the report would need to be completely rewritten and that, while it should retain its relevance for practitioners and those with a professional interest in the area, it should also contain advice of relevance to women and parents. At the same time, the Working Party was aware of a parallel piece of work, also arising from the Government response to the House of Commons Science and Technology Report on termination of pregnancy for fetal abnormality.[5] Much of that Working Party's report and, in particular, the conclusions and recommendations are of relevance to the issue of fetal awareness and, in this respect, the reports complement each other.

Particular acknowledgement is paid to those who took the lead in drafting the various chapters but responded constructively to discussion and modification, such that the report is one in which all of the participants contributed significantly. It is hoped that most will find the report helpful and that it goes some way to answering some of the criticisms of recent times, as well as offering sound advice to practitioners and consumers.

References

1. Royal College of Obstetricians and Gynaecologists. *Fetal Awareness: Report of a Working Party*. London: RCOG Press; 1997.
2. House of Commons Science and Technology Committee. *Scientific Developments Relating to the Abortion Act 1967: Twelfth Report of Session 2006–07. Volume I: Report, Together with Formal Minutes*. HC 1045-I. London: The Stationery Office; 2007 [www.publications.parliament.uk/pa/cm200607/cmselect/cmsctech/1045/1045i.pdf].
3. HM Government. *Government Response to the Report from the House of Commons Science and Technology Committee on the Scientific Developments Relating to the Abortion Act 1967*. Presented to Parliament by the Secretary of State for Health by Command of Her Majesty November 2007. Cm 7278. London: The Stationery Office; 2007 [www.official-documents.gov.uk/document/cm72/7278/7278.pdf].
4. Nuffield Council on Bioethics. Critical Care Decisions in Fetal and Neonatal Medicine: Ethical Issues. London: Nuffield Council on Bioethics; 2006 [www.nuffieldbioethics.org/fileLibrary/pdf/CCD_web_version_22_June_07_%28updated%29.pdf].
5. Royal College of Obstetricians and Gynaecologists. *Termination of Pregnancy for Fetal Abnormality: Report of a Working Party*. London: RCOG; 2010 [in press].

2. Neurobiological developments relevant to pain

This section examines current knowledge of central nervous system function during fetal and neonatal periods of human development. The aim is to provide a description of key events and changes to inform whether the fetus can reasonably be said to experience pain. To do this, we reviewed all new evidence related to the neurobiology of fetal pain that has been published in peer-reviewed journals listed on PubMed.

We begin by considering the scientific evidence for the presence of specific anatomical and physiological connections in the brain that are responsible for signalling noxious events to the central nervous system. Noxious stimuli are those that damage the tissues of the body or threaten to do so, such as surgical incision or physical trauma of the skin. In this context, we define pain as 'the unpleasant sensory or emotional response to such tissue damage' and trace the development of those responses through fetal development. We follow the path of the signals produced by tissue damage at sensory detectors in the skin and other organs, through to sensory circuits in the spinal cord, brainstem and thalamus and finally to the cerebral cortex, the site of higher level sensory processing. At each stage, we consider the scientific evidence for functional development and how this evidence may be interpreted. This section includes details derived from over 50 papers identified as relevant. Most were published since the last Working Party report[1] but this current report also considers the older material included in the previous report.

In addition to understanding the anatomical and physiological connections, it is also important to consider the psychological aspects of pain. Broadly accepted definitions of pain refer to pain as a subjective experience involving cognition, sensation and affective processes.[2] These psychological concepts are inevitably harder to address in a fetus but should not be ignored. A discussion of the importance of psychological processes in pain can be found in Box 1.

Development of neural pathways related to pain

The neural regions and pathways that are responsible for pain experience remain under debate but it is generally accepted that pain from physical trauma requires an intact pathway from the periphery, through the spinal cord, into the thalamus and on to regions of the cerebral cortex including the primary sensory cortex (S1), the insular cortex and the anterior cingulated cortex.[3,4] Fetal pain is not possible before these necessary neural pathways and structures (figure 1) have developed.

The generation of nerve signals from damaged tissue

For the fetus to respond to surgical damage, receptors in the affected tissue, such as skin and muscle, must signal the noxious stimulus or damage to the central nervous system. Nociceptors are sensory nerve terminals found in the skin and internal organs that convert tissue

damage into electrical signals. The pattern and strength of these nociceptor signals is the first determining step in generating pain. If nociceptor activity is prevented, such as following local anaesthesia, then pain is blocked. Deep tissue damage, for example, that cuts through nerve bundles causes a brief burst of electrical activity in some of the cut nerve endings known as an injury discharge.[5] The injured tissue, however, is now isolated from the central nervous system and, within a few minutes, the isolated tissue becomes 'numb' and pain free. Similarly, rare genetic defects that prevent all nociceptive signals result in a complete inability to sense pain.[6]

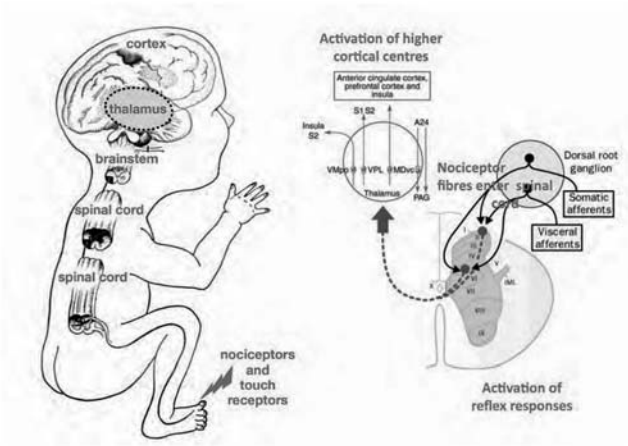

Figure 1. Pathways from the periphery through the spinal cord and into the thalamus and to the cortex. Nociceptor activity evoked by tissue damage reaches the spinal cord and can activate reflex responses through spinal cord connections. Pathways projecting to the thalamus and cortex may also be activated. Higher-level pain processing is thought to occur through a medial system (red lines) which has both ascending and descending components and a lateral system (blue lines) from the ventroposterior lateral (VPL) and ventromedial posterior (VMpo) nuclei. MDvc = mediodorsal ventral caudal nuclei; PAG = periaquaductal gray; S2 = secondary somatosensory cortex; S1 = primary somatosensory cortex; A24 = area 24, anterior cingulate cortex (adapted from Cervero and Laird,[55] Derbyshire[56] and Fitzgerald & Walker[57])

Anatomical studies of human fetal skin shows the presence of nerve terminals and fibres deep in the skin from 6 weeks of gestational age. These terminals are not nociceptors and are specialised for the processing of non-damaging sensations such as touch, vibration and temperature, rather than pain. From 10 weeks, nerve terminals become more numerous and extend towards the outer surface of the skin.[7,8] The terminals closer to the surface are likely to be immature nociceptors, necessary for pain experience following tissue damage, but they are not unequivocally present until 17 weeks.[8] In other mammals, newly formed fetal nociceptors are able to signal tissue damage but the intensity of their signals is weaker than in adults.[9] The internal organs develop nerve terminals later than the skin, beginning to appear from 13 weeks and then increasing and spreading with age, so that the pancreas, for example, is innervated by 20 weeks.[10]

Interpreting these data

Specialised nerve terminals, nociceptors, are likely to detect surgical tissue damage from early in fetal life (around 10 weeks for the skin and 13 weeks for the internal organs). These nociceptors gradually mature over the next 6–8 weeks and the strength of their signals increases over fetal life. The presence of nociceptors is necessary for perception of acute surgical pain and so pain is clearly not possible before the nociceptors first appear at 10 weeks. The presence of nociceptors alone, however, is not a sufficient condition for pain experience. The electrical activity that is generated at nociceptor terminals by tissue damage must also be conducted along nerve fibres from the skin and into the spinal cord and brain. It is only when the brain receives information about the damage that the fetus can have any potential of awareness of it.

The transmission of signals from damaged tissue to the lower levels of the central nervous system

Before any information about a noxious or tissue damaging stimulus can reach the brain, it has to be transmitted through the spinal cord (for the body) or the brainstem (for the head and neck). This transmission requires the growth of nerve fibres from the skin to the spinal cord or brainstem and then further growth of nerve fibres along the spinal cord or brainstem and into the brain. Staining of postmortem tissue reveals that nerve fibres grow into the fetal spinal cord from 8 weeks. These fibres, however, are specialised for the control of movement and some aspects of touching or prodding the body or positioning a limb.

The growth of nerve fibres connecting nociceptive terminals to the spinal cord lags behind that of other sensory inputs in non-human mammals. Similar connections in the human are also likely to lag but the specific timings remain unknown. Preliminary studies have failed to demonstrate nerve fibres from nociceptive terminals in the fetal post-mortem spinal cord before 19 weeks.[11]

The growth of sensory nerve fibres into the spinal cord is required for the fetus to display reflex movements in response to external stimuli. Sensory reflex responses are relatively simple, central nervous reactions to external events, some of which provide simple protection against damage. Examples of these reflexes include blinking in response to an air puff to the eye or the withdrawal of a limb in response to prodding the skin. The presence or absence of these reflexes at various stages of fetal life can provide information about the first functional sensory connections. In mammals these reflexes are mediated by the spinal cord and brainstem (Figure 1).

During the first 8 weeks of pregnancy, the human fetus displays a range of spontaneous movements, which are not actually reflexes, as they arise from random muscle actions rather than as reactions to a sensory stimulus. However, when sensory nerves have reached the skin, mechanical stimulation of the body can produce reflex movements. This confirms that these nerve fibres are carrying information about touch and have connected to the spinal cord and activated nerve fibres controlling motor actions. The fetal spinal cord and brainstem develop well before the cerebral cortex. This means that these reflex movements occur without any possibility of fetal awareness.

The exact timing of the first nociceptive reflex responses to more traumatic mechanical stimulation is not known but they are unlikely to occur before the second trimester, somewhat later than responses to touch. It is known that the fetus withdraws from a needle from about 18 weeks and also launches a stress response following needle puncture.[12] This stress response includes the release of hormones and neurotransmitters dependent on activity in areas of the midbrain. These findings confirm that signals about tissue damage are transmitted from the spinal cord and brainstem to the midbrain from at least 18 weeks.

Box 1. A discussion of the nature of pain

The word 'pain' is used in different ways. The most frequent use, especially with respect to subjects that cannot communicate verbally, is in describing the behavioural response to noxious stimulation. However, if we accept this use, we are presented with the difficulty of distinguishing between the responses of simple versus complex organisms. Fruit fly larvae, for example, have been demonstrated to bend and roll away when approached with a naked flame but most people would agree that larvae do not feel pain in the way that we do.

Ruling out the responses of larvae and similarly simple organisms as indicating pain is possible if we suggest that responses must include more than mere reflex responses to be labelled as a pain response. When someone reaches out and accidentally touches something very hot, there is an immediate tendency to drop the object. That reaction is entirely regulated by a simple loop of sensory neurons speaking to motor neurons in the spinal cord. Typically, the person will drop the object before there is any conscious appreciation of pain. The action of dropping the object indicates the presence of something noxious but does not necessarily indicate the presence of pain.

Most pain researchers adopt a definition of pain that emphasises the sensory, cognitive and affective response to a noxious event. This understanding of pain is supported by the International Association of Pain (IASP) which defines pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage…pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life'.[1] By this definition, pain does not have primacy over subjectivity, existing before and in addition to subjectivity, but is experienced through subjectivity. It suggests that pain is a part of knowledge and requires the existence of a conceptual apparatus that can marshal all its dimensions into a coherent experience.

Although there is considerable merit in the IASP definition of pain, it does tend towards a view of pain as being a constituent part of higher cognitive function. There is disquiet in denying a rawer, more primitive, form of pain or suffering that the fetus, neonate and many animals might experience.[2–4] One possible solution is to recognise that the newborn infant might be said to feel pain, whereas only the older infant can experience that they are in pain and explicitly share their condition with others as an acknowledged fact of being.[5]

Currently there is no immediately obvious way of resolving these arguments empirically. It is possible, however, to argue that even a raw sense of pain involves more than reflex activity and will, therefore, require the higher regions of the cortex to be connected and functional. The age when this minimum requirement is fulfilled is explored in the rest of this chapter.

References

1. Merskey H. The definition of pain. *Eur Psychiatry* 1991:6;153–9.
2. Anand KJS, Craig KD. New perspectives on the definition of pain. *Pain* 1996;67:3–6.
3. Lowery CL, Hardman MP, Manning N, Hall RW, Anand KJS. Neurodevelopmental changes of fetal pain. *Semin Perinatol* 2007;31:275–82.
4. Anand KJS. Consciousness, cortical function, and pain perception in non-verbal humans. *Behav Brain Sci* 2007;30:82–3.
5. Tallis R. *The Knowing Animal: A Philosophical Inquiry into Knowledge and Truth*. Edinburgh: Edinburgh University Press; 2005.

Interpreting these data

Observations of fetal movements in response to sensory stimulation show us that information about tissue stimulation has reached the spinal cord from 8 weeks. The demonstration of a hormonal stress response at 18 weeks following needle puncture shows us that information about tissue damage has reached the midbrain. A connection from the skin to the spinal cord and brain is a basic requirement for the fetus to feel or be aware of pain. Again, it is important to emphasise that, while such input to the spinal cord and brain is necessary for perception of acute surgical pain, it is not sufficient. Activity in the spinal cord, brainstem and subcortical midbrain structures are sufficient to generate reflexive behaviours and hormonal responses but are not sufficient to support pain awareness. At 18 weeks of gestational age, local spinal cord or brainstem reflexes control movement and, even as movement becomes more coordinated from 24 weeks, it does not require the involvement of higher brain centres. Extremely preterm infants of 24–30 weeks of gestation show the same motor responses to a noxious heel lance (required for clinical blood sampling) even when there is severe damage of the pathways connecting the spinal cord and brainstem to higher brain centres.[13] Also, such reactions to noxious stimuli, even those involving changes in facial expression, do not always correlate with cortical activity[14] when the nervous system is intact, showing that they cannot be assumed to reflect higher brain function.

Hormonal responses to needling show that there are functional brainstem and midbrain mediated reactions to noxious events but they, too, do not require higher brain processing to take place and can occur independently of sensory awareness. The specific relationship between pain and the release of hormones and neurotransmitters is unclear. In a prospective crossover study on 50 extremely low gestational age infants (less than 28 weeks of gestation), no difference in hormonal response was observed after heel lance[15] and, in adult mice, it is difficult to distinguish changes in levels of naturally occurring opioids due to stressful handling from those due to tissue damage.[16]

The transmission of signals from damaged tissue to cortical regions of the brain

Reflex movements and hormonal stress responses provide information about sensory connections at lower levels of the nervous system and cannot be assumed to indicate perception or awareness. For perception or awareness, the sensory information needs to be transmitted to the thalamus, the major subcortical sensory nucleus and then to the cortex, the highest region of the brain.

Anatomical evidence

At 8 weeks, the fetal brain is profoundly immature and its surface layer, the cerebral cortex, is smooth, with no indication of the folds (sulci and gyri) that are so prominent later.[17] There is also no internal cellular organisation in either the thalamus, which is the main source of sensory input to the cortex, or the cortex itself.[18,19] The limbic system, an evolutionary older part of the brain, consisting of interconnected deep brain structures involved in various fundamental drives and regulatory functions, is already discernable and has began to form interconnections.[20] The external surface of the brain is about 1 mm thick and consists of an inner and outer layer with no cortical plate, the structure that will gradually develop into the layers of the cortex proper.[21] At 13 weeks, a furrow or groove appears on each side of the brain,[17] which becomes part of the insular cortex around 15 weeks, a key region involved in the experience of external stimuli, including pain.[22] In spite of this, the fetal brain is still largely

smooth at 26 weeks. Massive growth of the brain after 34 weeks rapidly results in the characteristic folds and surface features of the more mature brain.

An important stage of cortical development is the formation of the subplate zone, a prominent, transient layer of the human fetal cerebral wall which develops around 13 weeks and gradually disappears after 32–34 weeks. The subplate is composed of newly arrived neurons and their connections together with other brain cells and cellular components and a large amount of extracellular material. All this makes the subplate very clearly distinguishable in fetal and neonatal brain scans (magnetic resonance images) and in postmortem brains. The subplate is thought to be the main synaptic or neuronal connection zone in the human fetal cortex where incoming fibres from the thalamus, the main sensory (and pain) relay centre, and other regions of the cortex gather during the crucial phase of cortical target area selection. Recent neurobiological evidence from other mammals shows that subplate is a site of spontaneous electrical activity and that this activity is required to build a framework for the precise organisation of cortical connections. The subplate is a focus of interest of paediatric neurology because damage to this area may lead to cognitive impairment in later life.[23]

The first projections to the subplate from the thalamus arrive between 12 and 18 weeks[21,24,25] and wait for the overlying cortical plate to mature and facilitate the invasion of neurons from the subplate.[26] Electrical activity arising from synaptic connections has been recorded in subplate neurons in isolated slices of mammalian brain but it is not known whether that activity can be selectively produced by thalamic connections or by noxious stimulation of body tissues in intact animals. It is known that this synaptic activity in the subplate performs a maturational function. In non-human mammals, synaptic activity in the subplate facilitates connections between thalamus and cortex and refines the early, initially crude, connections between the thalamus and cortex.[27]

By 24 weeks, substantial thalamocortical fibres have accumulated at the superficial edge of the subplate, which is the stepping-off point for axons growing towards their final cortical targets.[21] Between 24 and 32 weeks, there is substantial ingrowth of thalamocortical axons in the cortical plate of the frontal, somatosensory, visual and auditory cortex, and formation of the first synapses in the deep cortical plate. This is consistent with observations in neonates with rare brain malformations, such a lyssencephaly, where the brain resembles that of a fetus before 23–24 weeks of gestation, and which shows a lack of connections between the cortex and subcortical nuclei and an abnormal limbic system.[28]

At the same time, the relocation of neurons from the subplate to the cortical plate also begins around 24 weeks, thus coinciding with the invasion of thalamic afferents. This relocation is extremely rapid from about 34 weeks, leading to the dissolution of the subplate as the extracellular matrix and other growth-related and guidance molecules disappear.[21] The subplate has been observed to thin in the insula and in areas where cortical folding occurs rather earlier than the rest of the cortex, from at least 20 weeks.[29] It is currently uncertain whether this thinning is due to earlier maturation and potentially earlier synaptic activity in these regions, some of which are key areas in the experience of pain in adults,[3] or attributable to incidental morphological changes.

The arrival of thalamic fibres and formation of thalamocortical synapses in the newly formed cortex from 24 weeks onwards provides the minimum connection required for cortical processing of sensory events in the body. However, completion of the major pathways from the periphery to the cortex, at around 24 weeks, does not signal the end of cortical development but the beginning of a further maturational process. As spinothalamic pathways complete their connections with the cortex, they increasingly stimulate the development of intracortical pathways, which is the next major phase of neuronal maturation. Furthermore, the cortex sends

connections down to the brainstem and spinal cord; the motor centres of the brain have begun to form connections with the spinal cord and brainstem by 26–28 weeks.[30] This phase involves elaboration and refinement of neuron processes and connections, including selective elimination of some cell populations and corresponds to the cortical maturation described by Goldman-Rakic[31] in primates and by Chugani[32] in humans. McKinstry *et al.*[33] illustrated the effects of this development using diffusion tensor imaging in neonates born at 26 and 35 weeks. The proliferation of cortical neurons and the overgrowth of arborisation and synaptic contacts begins prenatally[32] but continues postnatally, together with synaptic elimination, pruning and programmed cell death.[31,32,34,35]

Physiological evidence

While the study of anatomical connections between brain regions provides important information about developing pain processes, the existence of a connection is not evidence of its function. Connections viewed under the microscope between the thalamus and the cortical plate at 24 weeks, for example, may or may not transmit information from nociceptors upon tissue damage. Fetal magnetoencephalography has been used to effectively record fetal auditory and visual evoked responses and spontaneous brain activity of cortical origin from 28 weeks and fetal brain activation to sound has been demonstrated using functional magnetic resonance imaging (fMRI) from 33 weeks. It has not been possible to record directly from human fetal cortex to establish when cortical neurons first begin to respond to tissue damaging inputs. Near infrared spectroscopy with preterm infants in intensive care, however, has demonstrated localised somatosensory cortical responses in premature newborn infants (from 24 weeks) following noxious heel lance[36] and venepuncture.[37] More recently, EEG has demonstrated a clear, time-locked, nociceptive-evoked potential in preterm infants following heel lance.[38] Thus, there is direct evidence of neural activity in primary sensory cortex following tissue damage in very premature infants equivalent to 24 weeks of gestational age.

Behavioural evidence

Fetal behavioural responses have also been used as indicators of stress or pain.[22,39] Shortly after the development of skin sensitivity, around 10 weeks, repeated stimulation results in hyperexcitability and a generalised movement of all limbs. After 26 weeks, this generalised movement gradually gives way to more coordinated behavioural responses that indicate improved organisation within the nervous system. Infants delivered at 26–31 weeks, for example, show coordinated facial expressions in response to heel prick,[23] although these are immature compared to older infants.[40] Four-D images of the fetus have also been reported to show fetuses 'scratching', 'smiling', 'crying' and 'sucking' at 26 weeks of gestational age.

Although these later behavioural responses are not spinal cord reflexes, the responses are still unlikely to involve higher cortical centres. An anencephalic fetus withdraws from noxious stimulation, demonstrating that this response is mediated at a subcortical level.[41] Similarly, infants with significant neonatal neurological injury due to a parenchymal brain injury respond to noxious stimulation with a pattern of behavioural reactions similar to infants without brain injury.[13]

Interpreting these data

The cortex is required for both the discriminative and emotional aspects of the processing of noxious stimuli and both anatomical and functional studies show that cortical neurons begin to receive input about sensory events in the body and the external environment from 24 weeks.

Long axonal tracts now course through the brain to the cortex and evoked responses in the primary sensory cortex indicate the presence of a spinothalamic connection and the ability of somatosensory cortical neurons to generate specific activity in response to tissue damaging stimulation. The primary sensory cortex is an important area in pain processing but it is only one of many areas that are active during pain experience.[4] Other important areas include the secondary somatosensory, the anterior cingulate and the insular cortices. Although we may speculate that these regions will also be functionally active from 24 weeks, similar to primary sensory cortex, there is no evidence for this at the moment.

It has been suggested that subcortical regions, including the brainstem, and transient brain structures, including the subplate, organise responses to noxious information at each stage of development and provide for a pain experience complete within itself at each stage.[42–44] There is, however, no evidence or rationale for subcortical and transient brain regions supporting mature function. Although developing brain circuits often display spontaneous neuronal activity this activity is a fundamental developmental process and not evidence of mature function.

The fact that the cortex can receive and process sensory inputs from 24 weeks is only the beginning of the story and does not necessarily mean that the fetus is aware of pain or knows that it is in pain. It is only after birth, when the development, organisation and reorganisation of the cortex occurs in relation to the action and reaction of the neonate and infant to a world of meaning and symbols, that the cortex can be assumed to have mature features. The cortex is an important step beyond the spinal cord and brainstem because it facilitates pain experience by enabling the higher functions of cognition, emotion and self-awareness that are realised in the postnatal environment. Thus, there is good evidence for claiming that the cortex is **necessary** for pain experience but not **sufficient**.

The interpretation of 4-D ultrasound images as evidence for emotional or sentient experience in the fetus is similarly problematic. While 4-D ultrasound provides better-quality images that can be useful to diagnose problems in fetal growth or structure, they provide no new evidence relevant to fetal sentience. As noted above, behavioural reactions can be mediated at a very low level in the brain and are not, therefore, evidence for experienced emotion or sentience. It is also important to recognise that 'labelling' a set of movements with a functional or emotional purpose can import too much certainty. Yawning, for example, is most likely a protective lung reflex that maintains proper lung inflation and prevents the developing alveoli (a kind of sponge-like material) from collapsing. While this protective reflex is unnecessary in the womb where oxygen is delivered by the umbilicus, it will be necessary soon after birth and therefore the neural connections that mediate it need to be fully functional well in advance of birth.

Sleep and wakefulness in the womb

It has been proposed that arguments around fetal pain can be resolved by the fact that the fetus never enters a state of wakefulness *in utero*.[45] This evidence is derived largely from observations of fetal lambs. Rigatto *et al*.,[46] for example, directly observed an unanaesthetised sheep fetus, *in utero*, through a Plexiglas window, for 5000 hours without observing signs of wakefulness such as eyes opening or coordinated movement of the head. Several factors explain this lack of wakefulness, including the environment of the womb, which is warm, buoyant and cushioned, and the presence of a chemical environment (most notably adenosine) that preserves a continuous sleep-like unconsciousness or sedation and suppresses higher cortical activation in the presence of intrusive external stimulation. Mellor *et al*.[45] also propose that the fetus is unconscious based on the presence of sleep-like EEG patterns observed in the lamb fetus, which enter a more quiescent state together with lack of movement, during hypoxic stress,[46,47] although it should be emphasised that this is quite different from the kind of nox-

ious stress generated by surgery discussed here. Mellor *et al.*[45] report that the general pattern of EEG during gestation is equivalent to a sleep-like state analogous to non-rapid eye movement and rapid eye movement sleep.

Interpreting these data

Although these data are derived from sheep, this species has been a useful investigative model of human pregnancy and the extrapolation of these data to the human fetus is plausible. Being asleep or awake is not as easy to distinguish in the fetus and newborn as it is in adults[48] but the broad categories can still be classified on the basis of EEG recordings. On this basis, sleep state differentiation appears in humans as early as 25 weeks in preterm infants and is complete at 30 weeks.[49] EEG recordings in late fetal baboons support these observations and define only two physiological states from EEG analysis, quiet sleep and active sleep.[50]

While the lack of fetal movement during anoxic stress in sheep may not be the same as the response to acute surgical tissue damage in humans, this work does highlight the important differences between fetal and neonatal life and the potential pitfalls of extrapolating from observations of newborn preterm infants to observations of the fetus. Sedation of the fetus and suppression of cortical arousal in times of stress imply that the cortex *in utero* responds differently from the neonatal cortex and that it is only after birth, with the separation of the baby from the uterus and the umbilical cord, that wakefulness truly begins. This conclusion is not inconsistent with reports of fetal conditioning and habituation to repeated exposure of sounds and smells in late pregnancy which are often referred to as fetal learning. Such responses do not require a cortex in a state of wakefulness and can be induced in simple circuits in lower organisms.[51]

Summary

Connections from the periphery to the cortex are not intact before 24 weeks of gestation. Most pain neuroscientists believe that the cortex is necessary for pain perception; cortical activation correlates strongly with pain experience and an absence of cortical activity generally indicates an absence of pain experience.[52–54] The lack of cortical connections before 24 weeks, therefore, implies that pain is not possible until after 24 weeks. Even after 24 weeks, there is continuing development and elaboration of intracortical networks. Furthermore, there is good evidence that the fetus is sedated by the physical environment of the womb and usually does not awaken before birth.

References

1. Royal College of Obstetricians and Gynaecologists. *Fetal Awareness: Report of a Working Party*. London: RCOG Press; 1997.
2. Merskey H. The definition of pain. *Eur Psychiatry* 1991:6;153–9.
3. Apkarian AV, Bushnell MC, Treede RD, Zubieta JK. Human brain mechanisms of pain perception and regulation in health and disease. *Eur J Pain* 2005;9:463–84.
4. Tracey I, Mantyh PW. The cerebral signature for pain perception and its modulation. *Neuron* 2007;55:377–91.
5. Blenk KH, Jänig W, Michaelis M, Vogel C. Prolonged injury discharge in unmyelinated nerve fibres following transection of the sural nerve in rats. *Neurosci Lett* 1996;215:185–8.
6. Cox JJ, Reimann F, Nicholas AK, Thornton G, Roberts E, Springell K, *et al.* An SCN9A channelopathy causes congenital inability to experience pain. *Nature* 2006;444:894–8.
7. Narisawa Y, Hashimoto K, Nihei Y, Pietruk T. Biological significance of dermal Merkel cells in development of cutaneous nerves in human fetal skin. *J Histochem Cytochem* 1992;40;65–71.

8. Terenghi G, Sundaresan M, Moscoso G, Polak JM. Neuropeptides and a neuronal marker in cutaneous innervation during human foetal development. *J Comp Neurol* 1993;328:595–603.
9. Koltzenburg M. The changing sensitivity in the life of the nociceptor. *Pain* 1999;Suppl 6:S93–102.
10. Amella C, Cappello F, Kahl P, Fritsch H, Lozanoff S, Sergi C. Spatial and temporal dynamics of innervation during the development of fetal human pancreas. *Neuroscience* 2008;154:1477–87.
11. Konstantinidou AD, Silos-Santiago I, Flaris N, Snider WD. Development of the primary afferent projection in human spinal cord. *J Comp Neurol* 1995;354:11–12.
12. Gitau R, Fisk NM, Glover V. Human fetal and maternal corticotrophin releasing hormone responses to acute stress. *Arch Dis Child Fetal Neonatal Ed* 2004;89:F29–32.
13. Oberlander TF, Grunau RE, Fitzgerald C, Whitfield MF. Does parenchymal brain injury affect biobehavioral pain responses in very low birth weight infants at 32 weeks' postconceptional age? *Pediatrics* 2002;110:570–6.
14. Slater R, Cantarella A, Franck L, Meek J, Fitzgerald M. How well do clinical pain assessment tools reflect pain in infants? *PLoS Med* 2008;5(6):e129.
15. Gibbins S, Stevens B, Beyene J, Chan PC, Bagg M, Asztalos E. Pain behaviours in extremely low gestational age infants. *Early Hum Dev* 2008;84:451–8.
16. Rasmussen NA, Farr LA. Beta-endorphin response to an acute pain stimulus. *J Neurosci Methods* 2009;177:285–8.
17. Afif A, Bouvier R, Buenerd A, Trouillas J, Mertens P. Development of the human fetal insular cortex: study of the gyration from 13 to 28 gestational weeks. *Brain Struct Funct* 2007;212:335–46.
18. Larroche JC. The marginal layer in the neocortex of a 7 week-old human embryo: a light and electron microscopic study. *Anat Embryol (Berl)* 1981;162:301–12.
19. Hevner RF. Development of connections in the human visual system during fetal mid-gestation: a DiI-tracing study. *J Neuropathol Exp Neurol* 2000;59:385–92.
20. Müller F, O'Rahilly R. The amygdaloid complex and the medial and lateral ventricular eminences in staged human embryos *J Anat* 2006;208:547–64.
21. Kostovi I, Jovanov-Milosevi N. The development of cerebral connections during the first 20–45 weeks' gestation. *Semin Fetal Neonat Med* 2006;11:415–22.
22. Craig AD. A new view of pain as a homeostatic emotion. *Trends Neurosci* 2003;26:303–7.
23. Kostovi I, Jovanov-Milosevi N. Subplate zone of the human brain: historical perspective and new concepts. *Coll Antropol* 2008;32 Suppl 1:3–8.
24. Kostovic I, Judas M. Correlation between the sequential ingrowth of afferents and transient patterns of cortical lamination in preterm infants. *Anat Rec* 2002;267:1–6.
25. Bystron I, Blakemore C, Rakic P. Development of the human cerebral cortex: Boulder Committee revisited. *Nat Rev Neurosci* 2008;9:110–22.
26. Molnar Z, Blakemore C. How do thalamic axons find their way to the cortex? *Trends Neurosci* 1995;18:389–97.
27. Kanold PO, Kara P, Reid RC, Shatz CJ. Role of subplate neurons in functional maturation of visual cortical columns. *Science* 2003;301:521–5.
28. Rollins N, Reyes T, Chia J. Diffusion tensor imaging in lissencephaly. *Am J Neuroradiol* 2005;26:1583–6.
29. Huang H, Zhang J, Wakana S, Zhang W, Ren T, Richards LJ, *et al*. White and gray matter development in human fetal, newborn and pediatric brains. *NeuroImage* 2006;33:27–38.
30. Eyre JA, Miller S, Clowry GJ, Conway EA, Watts C. Functional corticospinal projections are established prenatally in the human foetus permitting involvement in the development of spinal motor centres. *Brain* 2000;123:51–64.
31. Goldman-Rakic PS. Development of cortical circuitry and cognitive function. Child Development, 1987;58:601–22.
32. Chugani HT. Biological basis of emotions: brain systems and brain development. *Pediatrics* 1998;102:S1225–9.
33. McKinstry RC, Mathur A, Miller JH, Ozcan A, Snyder AZ, Schefft GL, *et al*. Radial organization of developing preterm human cerebral cortex revealed by non-invasive water diffusion anisotropy MRI. *Cereb Cortex* 2002;12:1237–43.
34. Huttenlocher PR, Dabholkar AS. Regional differences in synaptogenesis in human cerebral cortex. *J Comp Neurol* 1997;387:167–78.
35. Fitzgerald M. The development of nociceptive circuits. *Nat Rev Neurosci* 2005;6:507–20.
36. Slater R, Cantarella A, Gallella S, Worley A, Boyd S, Meek J, *et al*. Cortical pain responses in human infants. *J Neurosci* 2006;26:3662–6.

37. Bartocci M, Bergqvist LL, Lagercrantz H, Anand KJ. Pain activates cortical areas in the preterm newborn brain. *Pain* 2006;122:109–17.
38. Slater R, Worley A, Fabrizi L, Roberts S, Meek J, Boyd S, *et al.* Evoked potentials generated by noxious stimulation in the human infant brain. *Eur J Pain* 2009;May 28 [Epub ahead of print].
39. Anand KJS, Hickey PR. Pain and its effects in the human neonate and fetus. *N Engl J Med* 1987;317:1321–9.
40. Johnston CC, Stevens BJ, Franck LS, Jack A, Stremler R, Platt R. Factors explaining lack of response to heel stick in preterm newborns. *J Obstet Gynecol Neonat Nurs* 1999;28:587–94.
41. Visser GH, Laurini RN, de Vries JIP, Bekedam DJ, Prechtl HFR. Abnormal motor behaviour in anencephalic fetuses. *Early Hum Dev* 1985; 12:173–82.
42. Anand KJS, Craig KD. New perspectives on the definition of pain. *Pain* 1996;67:3–6.
43. Glover V, Fisk NM. Fetal pain: implications for research and practice. *Br J Obstet Gynaecol* 1999;106:881–6.
44. Lowery CL, Hardman MP, Manning N, Hall RW, Anand KJS. Neurodevelopmental changes of fetal pain. *Semin Perinatol* 2007;31:275–82.
45. Mellor DJ, Diesch TJ, Gunn AJ, Bennet L. The importance of 'awareness' for understanding fetal pain. Brain Research Reviews, 2005;49:455–71.
46. Rigatto H, Moore M, Cates D. Fetal breathing and behavior measured through a double-wall Plexiglas window in sheep. *J Appl Physiol* 1986;61:160–4.
47. Gunn AJ, Cook CJ, Williams CE, Johnston BM, Gluckman PD. Electrophysiological responses of the fetus to hypoxia and asphyxia. *J Dev Physiol* 1991;16:147–53.
48. Vecchierini MF, André M, d'Allest AM. Normal EEG of premature infants born between 24 and 30 weeks gestational age: terminology, definitions and maturation aspects. *Neurophysiol Clin* 2007;37:311–23.
49. Hunter CJ, Bennet L, Power GG, Roelfsema V, Blood AB, Quaedackers JS, *et al.* Key neuroprotective role for endogenous adenosine A1 receptor activation during asphyxia in the fetal sheep. *Stroke* 2003;34:2240–5.
50. Stark RI, Garland M, Daniel S, Myers MM. Diurnal rhythm of fetal behavioral state. *Sleep* 1998;21:167–76.
51. Hawkins RD, Kandel ER, Bailey CH. Molecular mechanisms of memory storage in Aplysia. *Biol Bull* 2006;210:174–91.
52. Fransson P, Skiöld B, Horsch S, Nordell A, Blennow M, Lagercrantz H, *et al.* Resting-state networks in the infant brain. *Proc Natl Acad Sci U S A* 104:15531–6.
53. Rosen SD, Camici PG. The brain-heart axis in the perception of cardiac pain: the elusive link between ischaemia and pain. *Ann Med* 2000;32:350–64.
54. Coghill RC, McHaffie JG, Yen YF. Neural correlates of interindividual differences in the subjective experience of pain. *Proc Natl Acad Sci U S A* 2003;100:8538–42.
55. Cervero F, Laird J. Visceral pain. *Lancet* 1999;353:2145–8
56. Derbyshire SWG. Measuring our natural painkiller. *Trends Neurosci* 2002;25:65–6.
57. Fitzgerald M, Walker SM. Infant pain management: a developmental neurobiological approach. *Nat Clin Pract Neurol* 2009;5:35–50.

14

3. Current clinical practice

Introduction

In the previous section we discussed the neurobiological basis and neuropsychological arguments around the possibility of fetal awareness of pain. Here, we focus upon the clinical perspective of fetal sensitivity to external stimuli *in utero* and the complex nature of the fetal stress response. Concerns have been raised that fetal medical procedures during pregnancy may lead not only to an immediate fetal stress response but also have long-term consequences. This section reviews all recent clinical developments to assess the validity of these concerns when balanced against the uncertain nature of the evidence for long-term harm, which has been based on postnatal rather than fetal studies, and the ubiquity of the fetal stress response, particularly during the normal process of vaginal birth.

Normal responses to vaginal delivery

Vaginal delivery may be considered a stress-inducing event to which most fetuses are subject. Fetuses born vaginally have higher levels of catecholamines, cortisol and endorphins than those born by elective caesarean section.[1,2] It is unclear whether this stress response is related to the painful stimulus of head compression or to other factors, such as mild hypoxaemia or maternal stress. In normal labour, this evidence of fetal stress would be considered a normal fetal physiological response and the stress is thought to have benefits for fetal survival. The labour-related surge in steroids and catecholamines is an important factor in activating sodium channels and promoting the clearance of lung fluid. Babies born by caesarean section before the onset of labour have an increased incidence of respiratory complications, such as transient tachypnoea of the newborn.[3] In addition, recent data show that elements of the stress response, perhaps noradrenaline or endorphins, have a short-term analgesic effect, so that babies born vaginally have an attenuated physiological and behavioural response to a painful stimulus compared with those born by elective caesarean section.[4] Evidence of endogenous fetal analgesia during vaginal birth, as well as the role of catecholamines in promoting lung fluid reabsorption and the respiratory depressant actions of fetal opiate exposure, all suggest that the current approach to intrapartum analgesia, centred around maternal, rather than fetal, requirements for pain relief, is the correct one. The evidence that stress responses during normal vaginal delivery have benefits cannot, however, be readily extrapolated to stress responses during pregnancy.

Fetal stress response

The fetal response to noxious stimuli, described in detail in section 2, comprises two elements, both of which need to be present for the fetus to feel pain. The first of these involves nociception and a physiological stress response to it, while the second requires cortical processing of the nociceptive stimulus to produce a negative emotional perception. The evidence clearly sug-

gests that the autonomic and endocrine pathways are in place for the fetus to mount a stress response as early as 18 weeks of gestation, with increases in cerebral blood flow, catecholamines and cortisol observed following invasive procedures.[5,6] These responses can be attenuated by administration of fetal analgesia at the start of the procedure.[7] It is worth noting that the fetal stress response can be elicited by a number of non-painful stimuli; the most extensively described is the response to acute hypoxia, where many of the components, such as increased cerebral blood flow, are part of a coordinated fetal response to minimise damage to organs such as the brain and heart. Increased cerebral blood flow, catecholamines and cortisol cannot therefore be interpreted as evidence that the fetus is feeling pain.

Data gathered from premature babies on intensive care units suggest that exposure to repeated, strong stimuli can alter cardiovascular responses to a painful stimulus later in infancy and that fetuses born with higher cortisol levels in cord blood, owing to vaginal delivery, have an altered stress response to vaccination. These data suggest that fetal exposure to 'stress' *in utero* can modulate the later function of the hypothalamic–pituitary axis. From this, it has been suggested that reducing the magnitude of the initial stress response, for example by using fetal analgesia, will have a beneficial effect. However, the degree to which these effects can be observed following fetal exposure to a painful stimulus remains uncertain, as the majority of studies to date are postnatal and refer to intense, repetitive stimuli that are not normally experienced *in utero*. The uncertain benefit of attenuating the fetal stress response to a noxious stimulus *in utero* by administering analgesia needs to be balanced against the practical difficulties to the administration of effective fetal analgesia, as well as the possibility of adverse effects.

Gestational age and fetal pain perception

In contrast to the endocrine and haemodynamic responses to a noxious stimulus, which are easily quantified, it has not been possible to directly measure the cortical response to such a stimulus. Assessments about the gestation at which a fetus could feel pain are therefore made on the basis of the existence of the necessary neural pathways for pain perception, particularly the nature of thalamocortical connections (see section 2), as well as indirect evidence for functionality based on evoked responses and evidence for a sleep–wake cycle of EEG activity. Interpretation of existing data indicates that cortical processing of pain perception, and therefore the ability of the fetus to feel pain, cannot occur before 24 weeks of gestation and that the nature of cortical activity becomes more complex as gestation advances from this point. It is reasonable to infer from this that the fetus does not require analgesia for interventions occurring before 24 weeks of gestation. Furthermore, and importantly, the evidence that analgesia confers any benefit on the fetus at any gestation is lacking.

Fetal exposure to noxious stimuli in utero

The fetus may be exposed to a variety of noxious stimuli *in utero*. The majority of fetuses will experience head compression owing to uterine contractions during labour, while a small number will have a needle placed in a blood vessel or organ. In addition, there is the vexed question as to whether the process of abortion represents a noxious stimulus to the fetus. In general, a noxious stimulus is considered to include forms of tissue damage related to physical interventions, such as head compression or needling, rather than fetal hypoxia or hypoglycaemia. A number of invasive procedures can be performed, as follows.

Most diagnostic procedures, including amniocentesis, chorion villus sampling and fetal blood sampling from the umbilical cord do not involve fetal contact. However, on occasion it is necessary to take a sample from the fetus itself, normally using a small gauge needle; for example, when fetal blood sampling from the umbilical vein in the fetal liver, when withdrawing fluid from a cyst or cystic organ or when carrying out a biopsy of fetal skin, liver, muscle, tumour or other tissue.

Again, the majority of therapeutic procedures, including fetal -ell or platelet transfusion via the umbilical cord and endoscopic laser ablation of twin–twin anastomoses on the placental surface, do not involve fetal contact. Some procedures, however, are performed directly on the fetus, including transfusion of donor red cells into the fetal intrahepatic umbilical vein or the peritoneal cavity. Also, drainage of abnormal fluid collections (for example, a dilated bladder or hydrothorax) can be achieved by a single aspiration using a needle or the percutaneous insertion of an indwelling shunt to the amniotic cavity. Similarly, endoscopic placement of a balloon that is inflated in the fetal trachea can be used to improve outcome in cases of congenital diaphragmatic hernia.

As mentioned previously, there is evidence that fetal needling results in a stress response and that this can be attenuated by administration of analgesia given directly to the fetus. In practice, maternal infusion of opiates has been used to sedate the fetus, to achieve immobilisation, rather than analgesia, just as muscle relaxants have been given directly to the fetus.

Open uterine surgery on the fetus is extremely unusual but has been described where surgical access to the fetus has been obtained during the second and third trimesters by performing a maternal hysterotomy. Fetal conditions treated via this approach include congenital diaphragmatic hernia and spina bifida. Use of these techniques is currently confined to a small number of specialist centres in the USA.

An *ex utero* intrapartum treatment can be performed if it is predicted that the fetal airway will be compromised at birth, normally as a result of a cervical tumour or laryngeal atresia. The fetus is partially delivered at the time of caesarean section and access obtained to the airway while the placental circulation maintains adequate oxygenation. As these procedures are performed under maternal general anaesthesia, the fetus is also anaesthetised as a result of transplacental passage of the high concentrations of volatile agents given to the mother.[8]

Administration of fetal analgesia

Lack of access to the fetus *in utero* limits ability to provide fetal analgesia. Two routes are available, either injection directly into the fetus or cord, or transplacental, following administration to the woman:

- direct fetal injection
- transplacental analgesia.

Direct fetal injection

Although it is possible to give an intramuscular or intravenous injection into the fetus under ultrasound guidance, there are a number of practical challenges to doing so:

- Fetal analgesia is not considered a sufficient indication to expose a pregnancy to the increased risk of miscarriage associated with insertion of a possible additional needle into the amniotic cavity. This means that the injection would have to be given as part of another diagnostic or therapeutic procedure involving the insertion of a needle.

- Giving an intramuscular injection before a diagnostic or therapeutic procedure will make the fetus move, with the potential of making the subsequent procedure more complicated.
- The majority of procedures involving percutaneous fetal needling are rapid, involving placing the needle appropriately, taking fluid or blood and then withdrawing the needle. There is normally insufficient time for the analgesic to work. It is important to minimise the time of intervention both for safety and to minimise exposure to the procedural stimulus.
- The needle and the trochar used for shunt placement is large (13 gauge) and not designed for intravascular access.

These considerations mean that the only procedure currently performed for which analgesia might be practical and appropriate is transfusion into the intrahepatic umbilical vein. This requires vascular access and the procedure can last for sufficient time (approximately 5–30 minutes) to allow analgesia time to have an effect.

Transplacental analgesia

Given to the woman, intravenously or via epidural, opiates such as morphine and fentanyl and benzodiazepines have all been shown to cross the placenta and have been associated with changes in fetal heart rate and neonatal respiratory depression.[6] Similarly, inhaled volatile anaesthetic gases such as isoflurane can cross the placenta. Indeed, when a woman is under general anaesthesia it is believed that the fetus is also anaesthetised. The fetus is more sensitive to the effects of anaesthetic agents and so fetal anaesthesia will normally be achieved.[8] In pregnant ewes, the dose of inhalational anaesthesia necessary to achieve maternal anaesthesia is sufficient for fetal anaesthesia.[9] However, in current obstetric practice maternal analgesia and anaesthesia is titrated against maternal requirements and physiological status rather than the status of the fetus. Lower concentrations in fetal compared with maternal blood mean that to achieve high fetal levels of an analgesic, such as morphine, the mother would be exposed to the risks of opiate overdose, including respiratory depression. These certainties outweigh uncertainty about the fetal need for analgesia.

Termination of pregnancy

A comprehensive evidence-based review of current UK practice is provided by the RCOG guideline, *The Care of Women Requesting Induced Abortion*.[10] A brief summary is provided here.

Surgical termination may be performed between 7 and 24 weeks of pregnancy, although procedures after 12 weeks should only be performed by a very experienced surgeon. In the UK, most centres perform surgical termination under general anaesthesia although at earlier gestations local anaesthesia with or without sedation is increasingly used. The procedure is often preceded by medical preparation of the cervix with prostaglandin administered around 3–6 hours earlier. This allows easier dilatation of the cervix in both parous and primigravid women and reduces blood loss, although in some cases the administration of prostaglandin 6 hours before evacuation will induce significant uterine activity, with associated pain and bleeding requiring the surgical procedure to be expedited. The pregnancy is removed by suction through a cannula and fetal death is very rapid. After 14 weeks, termination can be performed by dilatation and evacuation. For surgical termination in the UK, general anaesthesia is usually administered for dilatation and this will result in transfer of anaesthetic agents to the fetus. Al-

though fetal transfer occurs more slowly than maternal transfer, the amount of anaesthetic required is lower for the fetus and so fetal anaesthesia will normally be achieved.[8] However, as current evidence indicates the inability of the fetus to experience pain, certainly before the end of the second trimester, it should not be necessary to consider the need for fetal analgesia.

Hysterotomy (incision of the uterus) is rarely carried out, except where vaginal delivery is contraindicated because of placenta praevia or pelvic tumour or because of a fetal abnormality such as conjoined twins. This procedure is carried out under general anaesthesia with administration of substantially greater doses of anaesthetic and analgesic agents than is required for transcervical surgical termination of pregnancy, with consequently greater doses reaching the fetus.

Medical termination is induced by the administration of a prostaglandin, usually preceded by the administration of the antiprogesterone mifepristone. The regimen and dose vary according to gestation. At up to 9 weeks of amenorrhoea, the currently recommended regimen is oral mifepristone followed 24–48 hours later by misoprostol administered vaginally. Misoprostol can also be administered orally, sublingually or bucally, although the oral route is less effective and these routes are associated with more adverse effects. Between 9 and 12 weeks of gestation, a second dose of prostaglandin may be administered and occasionally further doses may be required. In the second trimester, a similar regimen of mifepristone followed by misoprostol, repeated as required, is used. The fetus is not directly manipulated during a medical termination of pregnancy. It will, however, be subjected to the compressive forces of uterine contractions. The likelihood of fetal death occurring during contractions or delivery, as a result of contraction related hypoxaemia, is higher at low gestations. Although women often receive analgesia and/or sedation during the procedure, this is for maternal benefit rather than fetal analgesia.

Feticide

When termination of pregnancy is performed after 22 weeks of gestation, it is recommended practice that feticide is performed before delivery, unless the fetal abnormality is lethal and will cause the death of the fetus during or immediately after delivery.[11] Although the rationale is to ensure fetal death at delivery, some parents may find it reassuring that the fetus will not experience any noxious stimuli during labour. Feticide can be used prior to medical termination of pregnancy for fetal abnormality after 22 weeks of gestation or for selective reduction of multiple pregnancies, either where one fetus has an abnormality or where the number of fetuses increases the risk of maternal morbidity or pregnancy complications to unacceptable levels.

The most common method of feticide is to place a small-gauge needle into the fetal heart under ultrasound guidance and inject 1–5 ml of strong potassium chloride (15%). This causes rapid asystole. Consideration can be also given to stopping fetal movements by the instillation of anaesthetic and/or muscle relaxant agents immediately before potassium chloride administration. The injection of digoxin into the amniotic fluid or into the fetus has also been used to bring about asystole.

Alternatively, if there is a possibility of vascular connection between twins (monochorionic and acardiac twins) and where it is necessary to achieve vascular isolation of the dead twin, feticide can be performed by occluding the umbilical circulation using diathermy applied by either bipolar diathermy forceps or unipolar diathermy at the fetal cord insertion. Multifetal reduction is usually performed in the late first or early second trimester, before 14 weeks of gestation, by injection of potassium chloride into the chest cavity or heart.

Summary

The implications for clinical practice of the neurobiological evidence presented in section 2 have been considered. Interpretation of existing data suggests that cortical processing and therefore fetal perception of pain cannot occur before 24 weeks of gestation. It is reasonable to infer from this that the fetus does not require analgesia for interventions occurring before 24 weeks of gestation. Diagnostic or therapeutic procedures that involve the fetus directly are very uncommon but do occur and can be associated with a stress response. However, this does not indicate that the fetus is aware or can feel pain. The case for administering analgesia before an invasive procedure (in addition to maternal general anaesthesia) after 24 weeks when the neuroanatomical connections are in place, needs to be considered together with the practicalities and risks of administration of fetal analgesia in continuing pregnancies and the uncertainties over long-term effects. Evidence that analgesia confers any benefit on the fetus at any gestation is lacking but should be a focus of future research that will need to include medium and longer-term as well as immediate outcomes. However, the need for maternal sedation before fetal interventions such as transfusion or feticide is still recognised, as it provides both maternal and procedural benefits.

References

57. Miller NM, Fisk NM, Modi N, Glover V. Stress responses at birth: determinants of cord arterial cortisol and links with cortisol response in infancy. *BJOG* 2005;112:921–6.

58. Vogl SE, Worda C, Egarter C, Bieglmayer C, Szekeres T, Huber J, *et al*. Mode of delivery is associated with maternal and fetal endocrine stress response. *BJOG* 2006;113:441–5.

59. Jain L, Eaton DC. Physiology of fetal lung fluid clearance and the effect of labor. Semin Perinatol 2006;30:34–43.

60. Bergqvist LL, Katz-Salamon M, Hertegård S, Anand KJ, Lagercrantz H. Mode of delivery modulates physiological and behavioral responses to neonatal pain. *J Perinatol* 2009;29:44–50.

61. Teixeira JM, Glover V, Fisk NM. Acute cerebral redistribution in response to invasive procedures in the human fetus. *Am J Obstet Gynecol* 1999;181:1018–25.

62. Lee SJ, Ralston HJP, Drey EA, Partridge JC, Rosen MA. Fetal pain: a systematic multidisciplinary review of the evidence. *J Am Med Assoc* 2005;294:947–54.

63. Fisk NM, Gitau R, Teixeira JM, Giannakoulopoulos X, Cameron AD, Glover VA. Effect of direct fetal opioid analgesia on fetal hormonal and hemodynamic stress response to intrauterine needling. *Anesthesiology* 2001;95:828–35.

64. De Buck, Deprest J, Van de Velde M. Anesthesia for fetal surgery. *Curr Opin Anaesthesiol* 2008;21:293–7.

65. Gregory GA, Wade JG, Beihl DR, Ong BY, Sitar DS. Fetal anesthetic requirement (MAC) for halothane. *Anesth Analg* 1983;62:9–14.

66. Royal College of Obstetricians and Gynaecologists. *Care of Women Requesting Induced Abortion*. London: RCOG Press; 2004.

67. Royal College of Obstetricians and Gynaecologists. *Further Issues Relating to Late Abortion, Fetal Viability and Registration of Births and Deaths*. RCOG Statement. London: RCOG; 2001.

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4. Information for women and parents

These questions and answers have been written to support women. They specifically relate to questions some women ask when having a termination of pregnancy, undergoing an invasive diagnostic procedure and about feticide. The questions below address issues to do with fetal awareness and pain only.

Note that each question and answer has been written to be as self-contained as possible unless specific sign-posting has been given. This is because women wanting information may not read all questions and answers.

Questions some women ask when having an abortion before 24 weeks

Will the fetus/baby feel pain?

No, the fetus does not experience pain. Pain relates to an unpleasant sensory or emotional response to tissue damage. To be aware of something or have pain, the body has to have developed special sensory structures and a joined-up nerve system between the brain and the rest of the body to communicate such a feeling. Although the framework for the nervous system in the growing fetus occurs early, it actually develops very slowly. Current research shows that the sensory structures are not developed or specialised enough to experience pain in a fetus less than 24 weeks.

After 24 weeks, it is difficult to say that the fetus experiences pain because this, like all other experiences, develops postnatally along with memory and other learned behaviours. In addition, increasing evidence suggests that the fetus never enters a state of wakefulness inside the womb. The placenta produces chemicals that suppress nervous system activity and awareness.

Will the process hurt the baby?

No. To be hurt, you need to feel pain. Current research shows that the sensory structures are not developed or specialised enough for a fetus to experience pain less than 24 weeks. Pain experience after 24 weeks depends upon a psychological development that is restricted before birth. See the question 'Will the fetus/baby feel pain?'

Will the fetus/baby be born alive?

The fetus will almost always die during the abortion process. This is always true for surgical termination. A fetus born before 22 weeks is not capable of surviving. If a medical abortion is carried out after 21 weeks and 6 days feticide will always be offered. To ensure that the baby is not born alive, the heart of the fetus will be stopped before the termination is carried out.

This involves an injection of a solution of potassium chloride directly into the fetal heart. A specially trained doctor carries out feticide. Before anything else is done, the fetal heart will be checked to ensure it has stopped.

When a late medical abortion is carried out and feticide is not performed, the fetus may show signs of life when delivered. This may involve body and limb movements. These movements are a reflex action. They cannot be avoided and can occur after death. This can be very distressing for both the woman and the clinical team looking after her, particularly if it is unexpected. Women undergoing late abortion should always be counselled about what might happen and should be aware of this possibility.

How does the fetus/baby die?

There are different methods of abortion. Which type of abortion you have depends on how many weeks pregnant you are. The different methods are:

- medical abortion – used most commonly in early and late abortions, this uses specific drugs to end the pregnancy
- Vacuum aspiration – used in early abortions where the contents of the womb are removed by suction
- Surgical dilatation and evacuation – used in later abortions where the fetus is removed in fragments.

Most abortions are carried out before the fetus has any chance of surviving outside the womb. In medical abortions, the fetus will usually die during the process and before delivery. Current research shows that the sensory structures are not developed or specialised enough to experience pain in a fetus of less than 24 weeks. If the abortion is carried out over 21 weeks and 6 days, feticide will be offered. This is where a specially trained doctor injects a solution of potassium chloride directly into the fetal heart to ensure it is not born alive. Fetal death is extremely quick.

Questions some women ask when undergoing an invasive diagnostic procedure

What harm could the procedure cause the baby?

To help to find out what problem the baby has, a practitioner has to carry out an invasive diagnostic procedure. This will involve inserting a needle into the uterus (womb) to take either a sample of fluid or tissue from the placenta or very occasionally from the umbilical cord. To ensure that the needle is inserted in the correct place, ultrasound guidance (a special device that uses sound waves to show the inside of the body to see organs and tissue) is used. All invasive procedures carry a small risk of miscarriage. Fewer than one woman in 100 (0.5–1%) will have a miscarriage because of the procedure.

Will the needle hurt the baby?

No. The procedure involves only the placenta or umbilical cord, which do not contain the nerves that are necessary to signal pain.

Does an anaesthetic or the pain relief I receive affect the baby?

If you are given a general anaesthetic for a diagnostic procedure, the substances used in this will cross the placenta to the baby. The effect will happen more slowly to the baby and will not cause any harm to the baby.

If you are given other forms of pain relief, there is evidence that they will cross the placenta to the baby, but the doses are not large enough to cause any harm.

Can the baby be given pain relief?

No. Current research shows that the sensory structures are not developed enough or specialised enough to respond to pain in a fetus of less than 24 weeks. See question on 'Will the fetus/baby feel pain?' In later pregnancy, when the fetus/baby is over 24 weeks, we do not yet have enough knowledge to know if providing pain relief would be beneficial. This means that it is extremely difficult to know what kind of pain relief should be used, how any pain relief should be given and whether it would be safe and effective. If pain relief was to reach the baby inside the womb, this would mean giving the mother larger and potentially dangerous doses to try and make sure enough crossed the placenta to the baby. This may cause more harm than benefit. Injecting pain relief drugs directly into the baby would increase the risk of miscarriage.

Questions some women ask when undergoing feticide

Will the baby suffer/feel pain?

No, the fetus does not experience pain. In addition, increasing evidence suggests that the fetus never enters a state of wakefulness inside the womb and that the placenta produces chemicals that suppress nervous system activity and awareness. Feticide is always offered when an abortion is carried out after 21 weeks and 6 days, unless the fetal abnormality is lethal and will cause death of the fetus during or immediately after delivery. A doctor who is specially trained in fetal medicine carries out feticide. To ensure the baby is not born alive, the doctor will inject a solution of potassium chloride directly into the fetal heart. Before anything else is done, the fetal heart will be checked to ensure it has stopped. Death is extremely quick after feticide.

How quickly will the baby die?

When feticide has been carried out, death is extremely quick.

A question some women ask when carrying a baby with a serious abnormality

Will the baby be in pain in the womb because of the condition that has been diagnosed?

This is very unlikely. Current research shows that the sensory structures are not developed or specialised enough to respond to pain in a fetus of less than 24 weeks. Even after 24 weeks it is difficult to say that the fetus experiences pain, because this, like all other experiences, develops postnatally along with memory and other learned behaviours. Moreover, the environment of the womb is usually protective with the fetus floating in the warm amniotic fluid.

5. Conclusions

The primary purpose of this report was to review current knowledge of the central nervous system to assess the likelihood that the fetus *in utero* could experience or be aware of pain. The experience of pain needs cognitive, sensory and affective components, as well as the necessary anatomical and physiological neural connections.

Nociceptors first appear at 10 weeks of gestation in the fetus but they are not sufficient for the experience of pain in themselves. That requires that electrical activity is conducted from the receptors into the spinal cord and to the brain. Fibres to nociceptor terminals in the spinal cord have not been demonstrated before 19 weeks of gestation, although it is known that the fetus withdraws from a needle and may exhibit a stress response from about 18 weeks. At this stage, it is apparent that activity in the spinal cord, brain stem and mid-brain structures are sufficient to generate reflex and humoral responses but not sufficient to support pain awareness. At the same time, completion of the major neural pathways from the periphery to the cortex, at around 24 weeks of gestation, heralds the beginning of a further neuronal maturation. The proliferation of cortical neurons and synaptic contacts begins prenatally but continues postnatally. Magnetic imaging techniques have recorded fetal auditory and visual responses from 28 weeks but it has not been possible to record directly when cortical neurons first begin to respond to tissue damaging inputs, although there is evidence of neural activity in primary sensory cortex in premature infants (around 24 weeks). It has been suggested that subcortical regions can organise responses to noxious stimuli and provide for the pain experience complete within itself but there is no evidence (or rationale) that the subcortical and transient brain regions support mature function.

Thus, although the cortex can process sensory input from 24 weeks, it does not mean that the fetus is aware of pain. There is sound evidence for claiming the cortex is necessary for pain experience but this is not to say that it is sufficient. Similarly, the interpretation of ultrasound images is problematic. It is important that 'labelling' a set of movements, such as 'yawning', with a functional or emotional purpose that is not possible does not imply such a purpose.

A further important feature is the suggestion, supported by increasing evidence, that the fetus never enters a state of wakefulness *in utero* and is bathed in a chemical environment that induces a sleep-like unconsciousness, suppressing higher cortical activation. Although this cannot be known with certainty, the observation highlights important differences between fetal and neonatal life and the potential pitfalls of extrapolating observations in newborn preterm infants to a fetus of the same gestational age.

From the clinical perspective, there is increasing awareness of the complex nature of the fetal response to stimuli *in utero* and a better understanding of the nature and circumstances of the stress response, including the likelihood of any short or long term consequences. These issues become particularly relevant when placed in the context of the normal processes involved in vaginal, or indeed caesarean, birth. Infants born vaginally demonstrate a chemical response to the birth processes that can be characterised as a stress response. This response can be provoked by a number of non-painful stimuli, such as hypoxia, but it is not clear that the response is merely that, rather than a physiological preparation for extra uterine life. Indeed, there is

even the possibility of a short-term analgesic effect during the birth process. What is clear, however, is that none of us has any memory of the pain of being born, which is not to say that birth, from the fetus' point of view, could not still have been a painful process.

A number of invasive procedures are required in the practice of fetal medicine, for both diagnostic and therapeutic purposes. Most involve needling of the cord or placenta, not the fetus itself. In some circumstances, a needle or catheter is inserted into the fetus or a biopsy is taken from the fetus. In these situations, it is likely that the procedure will be associated with a stress response in the fetus and the need for analgesia has been considered. Indeed, in the previous report, it was recommended that the use of analgesia be considered where the fetus was over 24 weeks of gestational age. However, this more recent review has concluded that the evidence that the fetus can and does experience pain is less compelling and accordingly the benefit of administering analgesia is less evident, while the risks and practicalities of so doing remain. So on the basis of 'first do no harm', prior to the procedures described in this report, analgesia is no longer considered necessary, from the perspective of fetal pain or awareness. However, it is recognised that maternal sedation confers both maternal and procedural benefits. Similarly, the need for analgesia before termination of pregnancy at advanced gestations, whether medical or surgical, is no longer considered necessary, although the need for feticide at viable or immediately previable gestations should still be considered.

These and related issues are considered in the revised Working Party report, *Termination of Pregnancy for Fetal Abnormality,* whose findings and recommendations supplement this report. Furthermore, consideration needs to be given to the education and support of clinical staff working in this difficult area.

Finally, an important addition in this report is the section on information for women and parents and it is hoped that this will provide helpful guidance as well as extending the relevance and usefulness of the report to a wider audience.

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Additional reading

Bruska M. An ultrastructural study of the myelination of the trigeminal ganglion in human foetuses aged 10 to 23 weeks. *Folia Morphologiica (Warszawa)* 2003;62:231–3.

Brusseau R, Myers L. Developing consciousness: fetal anesthesia and analgesia. *Seminars in Anesthesia, Perioperative Medicine and Pain* 2006;25:189–95.

Cervero F, Laird J. Visceral pain. *Lancet* 1999;353:2145–8.

Chalmers DJ. Facing up to the problem of consciousness. *Journal of Consciousness Studies* 1994;1:1–16.

Craig KD, Whitfield MF, Grunau RVE, Linton J, Hadjistavropoulos HD. Pain in the preterm neonate: behavioural and physiological indices. *Pain* 1993;52:287–99.

Derbyshire SWG. Measuring our natural painkiller. *Trends Neurosci* 2002;25:65–6.

Derbyshire SWG. Can fetuses feel pain? *BMJ* 2006;332,909–12.

de Weerd AW, van den Bossche AS. The development of sleep during the first months of life. *Sleep Med Rev* 2003;7:179–91.

Ellingson R. Variability of visually evoked responses in the human newborn. *Electroencephalogr Clin Neurophysiol* 1970;29:10–19.

Fitzgerald M. Neurobiology of fetal and neonatal pain. In: Wall P, Melzack R, editors. *Textbook of Pain*. Edinburgh: Churchill Livingstone; 1994. p. 153–63.

Fitzgerald M, Walker SM. Infant pain management: a developmental neurobiological approach. *Nat Clin Pract Neurol* 2009;5:35–50.

Foulkes T, Wood JN. Pain genes. *PLoS Genet* 2008;4:e1000086.

Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and -endorphin response to intrauterine needling. *Lancet* 1994;344:77–81.

Gitau R, Fisk NM, Teixeira JM, Cameron A, Glover V. Fetal hypothalamic-pituitary-adrenal stress responses to invasive procedures are independent of maternal responses. *J Clin Endocrinol Metab* 2001;86:104–9.

Gupta A, Giordano J. On the nature, assessment, and treatment of fetal pain: neurobiological bases, pragmatic issues, and ethical concerns. *Pain Physician* 2007;10:525–32.

Haynes J, Rees G. Decoding mental states from brain activity in humans. *Nat Rev Neurosci* 2006;7:523–34.

Hobson P. *The Cradle of Thought: Exploring the Origins of Thinking*. London: Macmillan; 2002.

Ismail KMK, Wilson M, Kilby MD. (2000). Fetal pain and analgesia. *Curr Obstet Gynaecol* 2000;10, 214–17.

Johnston CC, Stevens BJ. Experience in a neonatal intensive care unit affects pain response. *Pediatrics* 1996;98:925–30.

Merker B. Consciousness without a cerebral cortex: a challenge for neuroscience and medicine. *Behav Brain Sci* 2007;30:63–81.

Niedermeyer E. Maturation of the EEG: development of waking and sleep patterns. In: Niedermeyer E, Da Silva FL, editors. *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. 5th ed. Philadelphia: Lippincott, Williams and Wilkins; 2005. p. 209–34.

Nofzinger EA, Derbyshire SWG. Pain imaging in relation to sleep. In: Lavigne G, Sessle BJ, Choiniere M, Soja PJ, editors. *Sleep and Pain*. Seattle, WA: IASP Press; 2007. p. 153–73).

Rees G, Kreiman G, Koch C. Neural correlates of consciousness in humans. *Nat Rev Neurosci* 2002;3:261–70.

Rose DF, Eswaran H. Spontaneous neuronal activity in fetuses and newborns. Experimental Neurology, 2004;190:S37–43.

Smith RP, Gitau R, Glover V, Fisk NM. Pain and stress in the human fetus. *Eur J Obstet Gynecol Reprod Biol* 2000;92:161–5.

Ulfig N, Neudorfer F, Bohl J. Transient structures of the human fetal brain: subplate, thalamic reticular complex, ganglionic eminence. *Histol Histopathol* 2000;15:71–90.

Ven De Velde M, Jani J, De Buck F, Deprest J. Fetal pain perception and pain management. *Semin Fetal Neonat Med* 2006;11:232–6.

Vanhatalo S, van Nieuwenhuizen O. Fetal pain? *Brain Dev* 2000;22:145–50.

White MC, Wolf AR. Pain and stress in the human fetus. *Best Pract Res Clin Anaesthesiol* 2004;18:205–20.

Mr. FRANKS. You know, years ago there was a discussion about this issue taking place, and they put a picture of a 20-week baby up on the screen, and they asked the different participants there was it a baby, and it was amazing how the adults had to struggle with it. But one of the 2-year-olds in the audience, asked her, and she said, it is a baby.

I am always astonished how God seems to grant clarity and wisdom to 2-year-olds and seems to withhold it from some of the more sophisticated adults in the world.

I just appreciate the testimony here today, and I know it is a very emotional circumstance. Ms. Zink, I thank you for being here, thank you for telling us your story, and I wish you the very best in life. And I thank all of you for being here.

And without objection, all Members will have 5 legislative days to submit to the Chair additional written questions for the witnesses, which we will forward and ask the witnesses to respond to as promptly as they can so that their answers may be made a part of the record.

Without objection, all Members will have 5 legislative days with which to submit any additional materials for inclusion in the record.

With that, again, I thank the witnesses, and I thank the Members and observers, and this meeting, hearing is adjourned.

[Whereupon, at 5:12 p.m., the Subcommittee was adjourned.]

APPENDIX

MATERIAL SUBMITTED FOR THE HEARING RECORD

Prepared Statement of the Honorable Lamar Smith, a Representative in Congress from the State of Texas, and Chairman, Committee on the Judiciary

H.R. 3803, the "District of Columbia Pain-Capable Unborn Child Protection Act," was introduced by House Constitution Subcommittee Chairman Trent Franks and has over 180 cosponsors. The Senate companion version was introduced by Senator Mike Lee.

There are no restrictions on abortions until birth in the District of Columbia other than the federal law that bans partial-birth abortions. Yet since the Supreme Court's 1973 decision in *Roe v. Wade,* medical knowledge regarding the development of unborn babies and their capacities at various stages of growth has advanced dramatically.

The *New York Times* has explored research on the ability of unborn children to feel pain, noting the research of Kanwaljeet Anand, an Oxford- and Harvard-trained neonatal pediatrician. According to the *New York Times:*

> "As . . . technology improved, the preterm infants [Dr. Anand] cared for grew younger and younger and he noticed that even the most premature babies grimaced when pricked by a needle . . . [n]ew evidence, however, has persuaded him that fetuses can feel pain by 20 weeks gestation (that is, halfway through a full-term pregnancy) and possibly earlier."

In 2004, Dr. Anand took the stand in a courtroom to testify as an expert witness in the case of *Carhart v. Ashcroft*, one of the federal trials held to determine the constitutionality of the ban on partial-birth abortions.

When asked whether a fetus would feel pain during such a procedure, Dr. Anand answered "If the fetus is beyond 20 weeks of gestation . . . there will be pain caused to the fetus . . . And I believe it will be severe and excruciating pain."

Congress has the power to acknowledge these developments and enact H.R. 3803 under its authority over the District of Columbia, and prohibit abortions in D.C. after the point at which scientific evidence shows the unborn can feel pain, with some exceptions. Six states have already enacted the Pain-Capable Unborn Child Protection Act at the state level.

Those six state legislatures have adopted factual findings regarding the medical evidence that unborn children experience pain at least by 20 weeks after fertilization, about the start of the sixth month, and they prohibit abortions after that point, with narrowly drawn exceptions.

The Supreme Court has made clear that "The government may use its voice and its regulatory authority to show its profound respect for the life within the woman." And that Congress may show such respect for the unborn through "specific regulation because it implicates additional ethical and moral concerns that justify a special prohibition."

Further, there can be no doubt as to Congress' authority to legislate in the District of Columbia due to its exclusive authority under the District Clause. (This clause provides that Congress shall "exercise exclusive Legislation in all Cases whatsoever" over the District established as the seat of government of the United States, now known as the District of Columbia).

I thank Chairman Franks for his continuing leadership on this issue.

Material submitted by the Honorable Trent Franks, a Representative in Congress from the State of Arizona, and Chairman, Subcommittee on the Constitution

THE ETHICS & RELIGIOUS LIBERTY COMMISSION
OF THE SOUTHERN BAPTIST CONVENTION

Richard Land, D.Phil. *(Oxon.)*, President

July 18, 2012

The Honorable Trent Franks
U.S. House of Representatives
2435 Rayburn House Office Building
Washington, DC 20515

Dear Congressman Franks:

We write to thank you for your leadership in sponsoring the District of Columbia Pain-Capable Unborn Child Protection Act (H.R. 3803) to prohibit in the nation's capital the abortion of unborn babies who have reached 20 weeks post-fertilization or later, except to save the life of the mother. The Southern Baptist Ethics & Religious Liberty Commission enthusiastically supports this bill and urges your colleagues to do the same by cosponsoring the measure.

As you well know and as H.R. 3803 reports in its findings, strong scientific research demonstrates that by 20 weeks after fertilization—if not much earlier—unborn babies have the capacity to feel pain. By this stage of development, pain receptors are present throughout an unborn baby's body, with nerves connecting the receptors to the brain. It therefore comes as no surprise to us that, as medical studies have shown, when babies at this stage of development are subjected to stimuli that adults would recognize as painful, the unborn likewise react adversely, such as by recoiling.

Yet it is alarming that Congress, which has been granted legislative jurisdiction over the District of Columbia under Article I, Section 8 of the Constitution, allows this heinous practice of aborting pain-capable unborn children to continue in the nation's capital. This atrocious practice must be stopped.

We commend you for standing on the frontlines toward that goal. The District of Columbia Pain-Capable Unborn Child Protection Act is a much-needed response to the terrible human rights injustice of abortions of pain-capable babies in D.C. Please know of our commitment to stand with you in calling upon Congress to pass and President Obama to sign this bill into law this year.

Sincerely,

Richard D. Land

Main Office • 901 Commerce Street, Suite 550, Nashville, TN 37203 • *phone* 615.244.2495 • *fax* 615.242.0065
Leland House on Capitol Hill • 505 Second Street, N.E., Washington, D.C. 20002 • *phone* 202.547.8105 • *fax* 202.547.8165
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THE ETHICS & RELIGIOUS LIBERTY COMMISSION
OF THE SOUTHERN BAPTIST CONVENTION

Richard Land, D.Phil.(Oxon.), President

July 30, 2012

The Honorable John Boehner
Speaker of the House
U.S. House of Representatives
H-232, The Capitol
Washington, DC 20515

The Honorable Eric Cantor
House Majority Leader
U.S. House of Representatives
H-329, The Capitol
Washington, DC 20515

Dear Speaker Boehner and Majority Leader Cantor:

We write to thank you for scheduling a House vote on the District of Columbia Pain-Capable Unborn Child Protection Act (H.R. 3803), which would prohibit in the nation's capital the abortion of unborn babies who have reached 20 weeks post-fertilization or later, except to save the life of the mother. The Southern Baptist Ethics & Religious Liberty Commission enthusiastically supports this bill and urges all representatives to do the same by voting for the measure when it is considered on the House floor.

As you well know and as H.R. 3803 reports in its findings, strong scientific research demonstrates that by 20 weeks after fertilization—if not much earlier—unborn babies have the capacity to feel pain. By this stage of development, pain receptors are present throughout an unborn baby's body, with nerves connecting the receptors to the brain. It therefore comes as no surprise to us that, as medical studies have shown, when babies at this stage of development are subjected to stimuli that adults would recognize as painful, the unborn likewise react adversely, such as by recoiling.

Yet it is alarming that Congress, which has been granted legislative jurisdiction over the District of Columbia under Article I, Section 8 of the Constitution, allows this heinous practice of aborting pain-capable unborn children to continue in the nation's capital. This atrocious practice must be stopped.

The District of Columbia Pain-Capable Unborn Child Protection Act, sponsored by Rep. Trent Franks, is a much-needed response to the terrible human rights injustice of abortions of pain-capable babies in D.C. Thank you for making this bill a priority before the August recess. We urge all representatives to support enactment of H.R. 3803. Please know we will be notifying Southern Baptists on how representatives vote on the measure.

Sincerely,

Richard D. Land

cc: The Honorable Kevin McCarthy, House Majority Whip
The Honorable Trent Franks

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EXPERT REPORT OF KANWALJEET S. ANAND, M.B.B.S., D.Phil.

I am a pediatrician specialized in the care of critically ill newborns and children. For more than 20 years, I have conducted intensive research and study on the development of pain and stress in the human newborn and fetus. The U.S. Department of Justice has asked me to provide this expert report, describing the capacity of the fetus to feel pain and the effects of maternal anesthesia on that capacity, to assist the Court in its assessment of the Partial-Birth Abortion Ban Act of 2003.

Background and Qualifications

I received an M.B.B.S. (Bachelor of Medicine/Bachelor of Surgery, equivalent to an M.D.) from Mahatma Gandhi Memorial Medical College in Indore, India. After post-doctoral training in Pediatrics, I received a Rhodes Scholarship to study at the University of Oxford, England. For research performed at Oxford, on the hormonal and metabolic responses of premature and full-term newborns to the pain/stress caused by surgical operations and the effects of anesthesia in neonates, I received a D.Phil. (Doctor of Philosophy) from the Faculty of Medicine. Additional post-doctoral training was acquired in England, at Children's Hospital, Boston and at Massachusetts General Hospital, where I completed a fellowship in pediatric critical care medicine.

I have held academic appointments at the University of Oxford, Harvard Medical School, Emory University School of Medicine, and the University of Arkansas for Medical Sciences, where I served as Director of Critical Care Medicine in the Department of Pediatrics (1997-2003) and remain presently employed. I currently occupy the Morris & Hettie Oakley Endowed Chair in Pediatric Critical Care Medicine and serve as a tenured Professor of Pediatrics, Anesthesiology, Pharmacology and Neurobiology at the University of Arkansas for Medical Sciences. I serve as Director of the Pain Neurobiology Laboratory at Arkansas Children's Hospital Research Institute, where I study the effects of repetitive pain in early development. I am currently conducting a long-term study funded by the National Institutes of Health examining the effect of morphine on premature neonates from 23 to 32 weeks gestation. I also serve on the

Board of Directors of Arkansas Children's Hospital Research Institute. My clinical appointment at Arkansas Children's Hospital, as an Attending Physician, allows me to provide care for the patients admitted to the Pediatric Intensive Care Unit. I am a diplomate of the American Board of Pediatrics and the Sub-Board of Pediatric Critical Care Medicine, and licensed to practice medicine in the State of Arkansas. I have previously held medical licenses in Massachusetts, Georgia, in the United Kingdom and India.

I am the author or co-author of approximately 200 publications, and recipient of the Dr. Michael Blacow Award from the British Paediatric Association (1986), a Pediatric Resident Research Award from the American Academy of Pediatrics (1992), the first Young Investigator Award in Pediatric Pain from the International Association for the Study of Pain (1994), the Jeffrey Lawson Award from the American Pain Society (2000), and numerous other awards and honors. My research efforts have been focused on examining the immediate and long-term effects of pain in premature and full-term newborn infants, the development of a functional pain system during fetal and neonatal life, and the treatment of pain at these ages.

I am being compensated by the U.S. government at the rate of $450.00 per hour for my work on this case, plus the reimbursement of travel expenses.

During the past four years, I have testified as an expert witness in the following cases:

1. State of Texas vs. Kim Laird (pt. Michael Andrews); 9-24-2003 in Cass County Court, Texas.

2. State of Arkansas vs. Roshonda Smith (pt. Christian Cogshell); 11-4-2003 in Pulaski County Court, Jacksonville, Arkansas.

3. State of Arkansas vs. Efrem Burke (pt. Madison Crofford); Dec. 12-14, 2001 in Craighead County Court, Jonesboro, Arkansas.

4. Marilyn & Leon Espinoza vs. Morristown Memorial Hospital, S.E. Finch and others (pt. Alexandra Espinoza), Aug.-Sept., 2000 in Newark Federal Court, Newark, New Jersey.

Attached as Appendix A is my Curriculum Vitae, which lists in more detail my academic background, positions, research and publications. In forming the opinions contained in this Expert Report, I have considered the following materials, attached as Appendix B:

1. International Association for the Study of Pain; IASP Pain Terminology. A sample list of frequently used terms from: Classification of Chronic Pain, Second Edition, IASP Task Force on Taxonomy, edited by H. Merskey and N. Bogduk, IASP Press, Seattle, 1994, pp. 209-214. (Website: http://www.iasp-pain.org/terms-p.html)

2. Anand KJS, Hickey PR. Pain and its effects in the human neonate and fetus. New England Journal of Medicine (1987) 317:1321-1329.

3. Ward-Platt M, Anand KJS, Aynsley-Green A. Ontogeny of the stress response to surgery in the human fetus, neonate and child. Intensive Care Medicine (1989) 15:844-945.

4. Anand KJS, Craig KD. New perspectives on the definition of pain. Pain (1996) 67: 3-6.

5. Anand KJS, Rovnaghi C, Walden M, Churchill J. Consciousness, behavior, and clinical impact of the definition of pain. Pain Forum (1999) 8: 64-73.

6. Anand KJS, Maze M. Fentanyl, fetuses, and the stress response: signals from the beginnings of pain? Anesthesiology 2001; 95 (4): 823-825.

7. Bhutta AT, Anand KJS. Vulnerability of the developing brain: neuronal mechanisms. Clinics in Perinatology 2002; 29 (3): 357-372.

8. Anand KJS, Taylor B. Consciousness and the fetus. American Academy of Pediatrics: Biocthics Newsletter, Jan. 1999, pp.2-3.

9. Coskun V, Anand KJS. Development of supraspinal pain processing. In: Anand KJS, Stevens BJ, McGrath PJ, editors. Pain in Neonates. Vol. 10. Amsterdam: Elsevier Biomedical Publishers, 2000, pp. 23-54.

10. Modi N, Glover V. Fetal Pain and Stress. Chapter 11 in: Anand KJS, Stevens BJ, McGrath PJ (editors). Pain in Neonates, 2nd Edition, Elsevier Science Publishers, Amsterdam, 2000, pp. 217-228.

11. Hepper PG, Shahidullah S. The beginnings of mind--evidence from the behavior of the fetus. J Rep Infant Psyhol 1994; 12:143-54.

12. Molliver ME, Kostovic I, Loos Hvd. The development of synapses in cerebral cortex of the human fetus. Brain Research 1973; 50:403-7.

13. Smith RP, Gitau R, Glover V, Fisk NM. Pain and stress in the human fetus. European Journal of Obstetrics, Gynecology, & Reproductive Biology 2000; 92:161-5.

14. Partsch CJ, Sippell WG, MacKenzie IZ, Aynsley-Green A. The steroid hormonal milieu of the undisturbed human fetus and mother at 16-20 weeks gestation. Journal of Clinical Endocrinology & Metabolism 1991; 73:969-74.

15. Teixeira JM, Glover V, Fisk NM. Acute cerebral redistribution in response to invasive procedures in the human fetus. American Journal of Obstetrics & Gynecology 1999; 181:1018-25.

16. Fitzgerald M. Spontaneous and evoked activity of fetal primary afferents in vivo. Nature 1987; 326:603-5.

17. Kinney HC, Ottoson CK, White WF. Three-dimensional distribution of 3H-naloxone binding to opiate receptors in the human fetal and infant brainstem. Journal of Comparative Neurology 1990; 291:55-78.

18. Teixeira J, Fogliani R, Giannakoulopoulos X, Glover V, Fisk NM. Fetal haemodynamic stress response to invasive procedures. Lancet 1996; 347:624.

19. Kopecky EA, Ryan ML, Barrett JF, et al. Fetal response to maternally administered morphine. American Journal of Obstetrics & Gynecology 2000; 183:424-30.

20. Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and beta-endorphin response to intrauterine needling. Lancet 1994; 344:77-81.

21. Gitau R, Fisk NM, Teixeira JM, Cameron A, Glover V. Fetal hypothalamic-pituitary-adrenal stress responses to invasive procedures are independent of maternal responses. Journal of Clinical Endocrinology & Metabolism 2001; 86:104-9.

22. Vanhatalo S, van Nieuwenhuizen O. Fetal pain? Brain & Development 2000; 22:145-50.

23. Fisk NM, Gitau R, Teixeira JM, Giannakoulopoulos X, Cameron AD, Glover VA. Effect of direct fetal opioid analgesia on fetal hormonal and haemodynamic stress response to intrauterine needling. Anesthesiology 2001; 95:828-835.

24. Saunders PJ. Do fetuses feel pain? We should give them the benefit of the doubt. British Medical Journal 1997; 314:303.

25. Giannakoulopoulos X, Teixeira J, Fisk N, Glover V. Human fetal and maternal noradrenaline responses to invasive procedures. Pediatric Research 1999; 45:494-9.

26. Goldman-Rakic PS. Development of cortical circuitry and cognitive function. Child Development 1987; 58:601-22.

27. Craig AD. A new view of Pain as a Homeostatic Emotion. Trends in Neurosciences 2003; 26 (6): 303-307.

Summary of Opinion

It is my opinion that the human fetus possesses the ability to experience pain from 20 weeks of gestation, if not earlier, and the pain perceived by a fetus is possibly more intense than that perceived by term newborns or older children. The process of (a) grasping the lower extremity of the fetus with a forceps or other surgical instrument, (b) manipulating or rotating the fetal position within the uterus, (c) forcible extraction of the fetal legs and lower body through the uterine cervix, (d) surgical incision of the fetal cranium/upper neck area of the fetus, and (e) entrance into the cranial vault (followed by vacuum suctioning of the fetal brain) during an abortion procedure will result in prolonged and intense pain experienced by the human fetus, if that fetus is at or beyond the neurological maturity associated with 20 weeks of gestation. Anesthetic agents that are routinely administered to the mother during this procedure would be insufficient to ensure that the fetus does not feel pain, and higher doses of anesthetic drugs, enough to produce fetal anesthesia, would seriously compromise the health of the mother. Thus, it is my opinion that the fetus would be subjected to intense pain, occurring prior to fetal demise, from the abortion procedures described in the Partial-Birth Abortion Ban Act of 2003.

The Capacity of the Fetus to Experience Pain

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment." The human fetus is obviously incapable of verbal expression and, therefore, the evidence for fetal pain must be based on surrogate markers, including anatomical, functional, physiological and behavioral indicators that are correlated with pain, from studies of pain in children or adults. Multiple lines of scientific evidence converge to support the conclusion that the human fetus can experience pain from 20 weeks of gestation, and possibly as early as 16 weeks of gestation.

Anatomical Development:

The neural pathways for pain include sensory receptors in the skin connected to nerve fibers, which lead to pain processing in the dorsal horn of the spinal cord. Nerve tracts from these spinal cord areas transmit the signals of pain to supraspinal centers located primarily in the brainstem, thalamus, and cerebral cortex of the brain.

Fully-functioning sensory receptors appear in the skin around the mouth of the fetus at 7 weeks and spread to all skin and mucous surfaces before 20 weeks of gestation. Nerve fibers precede the appearance of these skin receptors, and are capable of transmitting sensory stimuli from the periphery to the spinal cord at all times. Until the maturation of connections between unmyelinated pain-specific fibers and spinal cord neurons is complete, pain impulses are transmitted by a population of nerve fibers that only carry the touch sensation in later life. Dorsal horn neurons in the spinal cord begin to develop in the first trimester (before 13 weeks), with increasing anatomical complexity and functional maturation throughout fetal life. The pattern of functional maturation is such that incoming painful impulses are readily transmitted to the brain, but modulation or inhibition of these impulses does not develop until late gestation (36 to 40 weeks) or even 6-8 weeks after birth.

The architectonic organization and differentiation of the neuronal cell types in the fetal brainstem (including the medulla, pons, and midbrain) and fetal thalamus occurs during the first and second trimesters of pregnancy. Transient developmental characteristics appear during early maturation in these areas; for example, the reticular thalamic nucleus plays a major role in the fetal brain, but is not visible in the adult brain. Cellular development in these areas reveals highly diverse, bipolar, multipolar or polymorphous, transmitter-reactive neurons, with highly elaborate branching of dendrites during fetal development. Specific molecular markers in these neurons are correlated with the functional receptors, chemical transmitters, and enzymes that are expressed in the adult human brain. These diverse neuronal types, their elaborate dendrites and axons, as well as their neurochemical development imply a functional role in early development. The brainstem and thalamic areas serve as intermediate targets for the sensory axons growing centrally from different levels of the spinal cord, which are sorted and directed towards different cortical and sub-cortical targets.

The imaging of glucose metabolic rates in the neonatal brain shows the highest functional activity in the thalamus and brain stem, in addition to sensory cortical areas. Magnetic resonance imaging (MRI) scans also show that the earliest myelination occurs in the posterior brainstem and the ventrolateral nuclei of the thalamus, which are the areas associated with pain processing during fetal development.

The cerebral cortex starts to form at about 8-10 weeks of human gestation, although early cortical neurons have few axonal or dendritic connections. Maturation and differentiation of these neurons occurs in the second trimester and the sub-plate zone is formed at around 15 weeks. Massive increases in dendritic arborization and synaptogenesis begin at 18-20 weeks of gestation, with sub-plate neurons serving as a signaling station for axonal connections from the sub-cortical areas. The fetal neocortex is penetrated by the fibers from sensory thalamic nuclei by 20 weeks, whereas other fibers (not routed through the thalamus) have penetrated the sub-plate zone by 13 weeks and reached the cortical plate by 16 weeks of gestation, providing the final anatomical link for inputs to reach the developing cortex. Structural data for fetal brains at 17-40 weeks of gestation showed that cortical layer thickness increases linearly with age, while the number of cortical neurons (corrected for surface and gyral growth) increases 10-fold from 12 to 28 weeks, reaching a peak at 28 to 32 weeks. Cortical columns (functional units of the cerebral cortex) increase in the fetal sensory cortex; the number of dendritic connections varies with age and the body-map representation for each column, which may provide a structural basis for the relationships between stimulus intensity and perception. Numerous studies show that the time course of developmental gene expression critically depends on afferent (sensory) activity entering the cortex. Thus, "neurons that fire together wire together" or activity-dependent effects on gene expression lead to the establishment of cortical maps during development.

Physiological Responses:

Fetuses have been observed to exhibit hormonal stress responses to painful stimuli from as early as 16 weeks of gestation, which provide additional evidence that the fetus can experience pain. Studies have demonstrated that certain stress hormones (plasma cortisol, catecholamines and β-endorphin) increased significantly in fetuses given blood transfusions through a needle

placed, under ultrasound guidance, in the intra-hepatic vein (reached by piercing the fetus's abdominal wall), whereas no consistent responses occurred in the fetuses transfused via a needle placed at the insertion of the umbilical cord (which is not innervated). The magnitude of the stress hormone responses was correlated with the duration of the painful stimulation. In addition, these hormonal responses were reduced when fentanyl (a pain-relieving opiate drug) was administered directly to the fetus.

Other studies have examined the redistribution of blood flow within the fetus caused by invasive procedures such as fetal blood sampling, body cavity aspirations, and insertion of feto-amniotic shunts. These studies revealed that the blood flow to the brain decreased within 70 seconds after painful stimulation in fetuses from as early as 16 weeks of gestation. Hormonal or circulatory responses from the fetus may not vouchsafe conscious pain perception, although their absence would be more likely if sensory stimuli from these invasive procedures were not reaching the thalamus and hypothalamus.

Increased Sensitivity to Pain in the Fetus:

The highest density of pain receptors per square inch of skin in human development occurs *in utero* from 20 to 30 weeks gestation. During this period, the epidermis is still very thin, leaving nerve fibers closer to the surface of the skin than in older neonates and adults. Even though the fetus possesses excitatory pain mechanisms (receptors and fibers that recognize and respond to painful stimuli) before 20 weeks of gestation, the pain inhibitory mechanisms (fibers which dampen and modulate the experience of pain) do not begin to develop until 32-34 weeks of gestation. Thus, a fetus at 20 to 32 weeks of gestation would experience a much more intense pain than older infants or children or adults, when these age groups are subjected to similar types of injury or handling. Other mechanisms supporting an increased sensitivity to pain during fetal life are reviewed in the accompanying materials (Appendix B).

The Question of Fetal Consciousness:

More than 3 decades of research shows that preterm infants are actively perceiving, learning, and organizing information, and are constantly striving to regulate themselves, their

environment and their experiences. All preterm infants actively approach and favor experiences that are developmentally supportive and actively avoid experiences that are developmentally disruptive. These behaviors are designed to support the conservation of energy, the organization of sleep-wake cycles, and the achievement of successive, age-related developmental milestones.

If preterm neonates from 23 weeks can respond to and organize their experiences, it is likely that rudimentary forms of these abilities are present *in utero*, which raises the question of fetal consciousness. Consciousness is associated with shifting patterns of activity of the cerebral cortex, but its mechanisms are not completely understood even in the adult brain. Thus, it may not be possible to obtain unequivocal evidence for fetal consciousness. A British Commission of Inquiry into Fetal Sentience declared that fetuses may be conscious from six weeks of gestation, whereas a committee from the Royal College of Obstetrics and Gynaecology countered that fetuses cannot be sentient before 26 weeks of gestation.

If cortical activity is considered as a marker for fetal consciousness, the electroencephalogram (EEG) signals such activity from 19 to 20 weeks of gestation and sustained EEGs can be recorded from fetuses of 23 weeks gestation. From about 20 weeks, fetuses start responding to light, sound, touch and taste, with progressive increases in the complexity of their spontaneous movements at this time. Somatosensory evoked potentials can be recorded from the sensory cortex after 24 weeks of gestation.

Similar to the physiological responses of preterm neonates, fetuses greater than 16-20 weeks respond to painful procedures with hormonal stress responses, noted from changes in plasma cortisol, catecholamines and β-endorphin, and from changes in the pulsatility index of the middle cerebral artery within 70 seconds after stimulation. Experimental findings show that human fetuses can acquire distinct verbal memories from prenatal experiences (studied only in the third trimester of pregnancy), which supports the concept that consciousness appears before birth. All the lines of evidence reviewed above suggest the presence of consciousness from about 20-22 weeks of fetal life.

The Effect of Maternal Anesthesia on the Fetus

The effect of maternal anesthesia on the fetus' capacity to experience pain depends on the type of anesthetic, the dosage given, and the method of administration. To reach the fetus, a drug administered to the mother would have to avoid metabolism by the maternal liver, enter the maternal bloodstream, cross the placental membrane, reach the fetal circulation in sufficient concentrations, and cross the fetal blood/brain barrier to produce significant clinical effects on the fetus. Methods that are routinely applied, for example, a pudendal nerve block, epidural anesthesia, or other methods of local/regional anesthesia would provide no protection against pain to the fetus. General anesthetics (inhalational anesthetics and certain opiates, such as fentanyl and sufentanyl) can provide some degree of pain relief to the fetus, because they readily cross the placental barrier and fetal blood/brain barrier. Nevertheless, studies of drug efficacy using anesthetic agents show that the fetus would require a higher concentration of anesthetics in the fetal circulation to achieve the same clinical anesthetic effects as occurring on the mother. Thus, doses of anesthesia that would be toxic to the mother will be required to ensure that the fetus experiences no pain during a surgical procedure.

Dated: January 15, 2004

KANWALJEET S. ANAND, M.B.B.S., D.Phil.

national RIGHT TO LIFE committee, inc.

512 10th Street, NW Washington, DC 20004-1401
(202) 626-8800 FAX: (202) 737-9189 Website: www.nrlc.org

To: Science and Medicine Editors/Reporters

From: Douglas Johnson, NRLC Legislative Director
(202) 626-8820, fax (202) 347-3668

Re: medical pseudo-science that endangers women and their babies

Date: Tuesday, January 2, 1996

As part of a campaign against a bill pending in Congress, certain advocacy groups have disseminated a medical claim that has "absolutely no basis in scientific fact," according to the physicians'-specialty group with expertise on the matter. Moreover, says the physicians' group, this pseudo-scientific claim has itself become so widely disseminated through the media that it now poses a danger to the health of pregnant women and their babies.

It's a story that so far has gone virtually uncovered by the mainstream press-- although it is the subject of an article in the January 1 edition of *American Medical News*, the official newspaper of the American Medical Association (enclosed).

The bogus claim is this: anesthesia, given to a pregnant woman, kills the fetus/baby, prior to the performance of a late-term abortion.

This claim was invented last summer by certain opponents of a bill pending in Congress to ban the partial-birth abortion procedure. [In this procedure, a living fetus/baby (4½ to 9 months) is pulled feet-first from the womb, except for the head; the back of the skull is punctured, and the brain suctioned out.[1]]

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[1]Detailed documentation on the partial-birth abortion method and the reasons why it is performed-- much of it drawn from the writings of practitioners-- is available on request from NRLC. The enclosed drawings have been validated as medically accurate by experts on both sides of the abortion debate.

Specifically, certain opponents of the bill have argued as follows: (a) anesthesia given to the mother kills the fetus/baby before the rest of the abortion procedure, therefore (b) it is misleading to call the procedure a "partial birth," and (c) any concerns that the fetus/baby experiences great pain during the partial-birth abortion procedure are misplaced.

However, the American Society of Anesthesiologists (ASA) recently became so distressed by these claims that the ASA requested the opportunity to testify before the Senate Judiciary Committee. In its testimony, the ASA said that (a) the claim that anesthesia kills a fetus/baby has "absolutely no basis in scientific fact," and (b) the claim is "misleading and potentially dangerous" to pregnant women, since it may deter them from consenting to be anesthetized for medically necessary procedures for fear of harming their babies.

Dr. Norig Ellison, president of the American Society of Anesthesiologists, said that ***regional*** **(local)** anesthesia has no effect on the fetus.[2] Some ***general*** anesthetics reach the fetus in levels less than in the mother, but they do the baby no harm-- and indeed, they "will provide no-to-little analgesia [protection from pain] to the fetus," Dr. Ellison said.

The January 1 *American Medical News* article quotes Dr. David Birnbach, vice-president of the Society for Obstetric Anesthesia and Perinatology, as referring to the fetal-death claim as "crazy." Even at the extraordinarily high doses of anesthesia that (it is now claimed) the late abortionist Dr. James McMahon utilized, "anesthesia does not kill an infant if you don't kill the mother," Dr. Birnbach said.

Anesthesiologists stress that the continued dissemination of this misinformation is jeopardizing the health and lives of pregnant women and their babies in contexts entirely unrelated to abortion. Dr. Ellison testified:

> I am deeply concerned. . . that widespread publicity [given to this claim]...may cause pregnant women to delay necessary and perhaps life-saving medical procedures, totally unrelated to the birthing process, due to misinformation regarding the effect of anesthetics on the fetus. [Testimony before Senate Judiciary Committee, Nov. 17, 1995]

The *American Medical News* article makes the same point:

---

[2] The best known practitioner of partial-birth abortions, Dr. Martin Haskell of Dayton, Ohio, performs these procedures "under *local* anesthesia," according to his 1992 paper "Dilation and Extraction for Late Second Trimester Abortion," in which he explains step-by-step how to perform the entire procedure. Dr. Haskell has also acknowledged, in a tape-recorded 1993 interview with *American Medical News*, that most of the fetuses are alive at the time that he removes them from the womb-- and that "80%" of these procedures, in his practice, are "purely elective."

> **Medical experts contend the claim is scientifically unsound and irresponsible, unnecessarily worrying pregnant women who need anesthesia.... In fact, cases of maternal concern have already surfaced. Dr. Birnbach said he has already had patients raise questions. And Rep. Tom Coburn, MD, an Oklahoma Republican who still delivers babies when he goes home on weekends, said he just had a patient refuse epidural anesthesia during childbirth after hearing those claims.**

Despite the authoritative statements by the ASA and other experts, some prominent opponents of the bill continue to propagate the myth that anesthesia kills unborn babies. Indeed, the myth has taken on a life of its own, and it continues to spread in ever-widening circles. (See Addendum for some examples.)

The issue raised by ASA's warning really has nothing to do with the merits or demerits of the abortion bill itself (regarding which the ASA has no position). Regardless of the merits of the bill, the wide dissemination of gross misinformation regarding the effects of anesthesia on a human fetus/unborn baby is a disservice to the public, and needs to be corrected.

Original source documents for statements quoted in this memo, and related documentation, are available on request from NRLC, (202) 626-8820, fax (202) 347-3668, e-mail Legfederal@aol.com.

[The *American Medical News* article "Anesthesiologists Question Claims in Abortion Debate" (January 1, 1995) is attached. Other documentation is available on request, including:

Written testimony of Dr. Norig Ellison, president, American Society of Anesthesiologists before the Senate Judiciary Committee, Nov. 17, 1995

Transcript of exchange among Dr. Ellison, Dr. Mary Campbell of Planned Parenthood, and Sen. Spence Abraham (R-Mi.), Senate Judiciary Committee, Nov. 17, 1995

Letter from Dr. Norig Ellison to Senate Judiciary Committee, Nov. 22, 1995]

ADDENDUM: EXAMPLES OF VECTORS FOR THE "ANESTHESIA MYTH"

Far from dying out, the "anesthesia myth" continues to be disseminated to ever-wider audiences by advocates, editorial boards, reporters, and others. A few examples follow; many others could be cited.

- On December 15, the *New York Daily News* (circulation 725,000) ran an editorial defending partial-birth abortions, which said:

> The fetus is partially removed from the womb, its head collapsed and brain suctioned out so it will fit through the birth canal. The anesthesia given to the woman kills the fetus before the full procedure takes place. But you won't hear that from the anti-abortion extreme. It would have everybody believe the fetus is dragged alive from the womb of a woman just weeks away from birth. Not true.

- One of the leading proponents (to this day) of the "anesthesia myth" is Kate Michelman, president of the National Abortion and Reproductive Rights Action League (NARAL). For example, in an interview on "Newsmakers," KMOX-AM in St. Louis on Nov. 2, Ms. Michelman said:

> The other side grossly distorted the procedure. There is no such thing as a 'partial-birth'. That's, that's a term made up by people like these anti-choice folks that you had on the radio. The fetus-- I mean, it is a termination of the fetal life, there's no question about that. **And the fetus, is, before the procedure begins, the anesthesia that they give the woman already causes the demise of the fetus. That is, it is not true that they're born partially. That is a gross distortion, and it's really a disservice to the public to say this.**

Here are a few other examples:

- Syndicated columnist Ellen Goodman wrote in mid-November that, if one relied on statements by supporters of the bill, "You wouldn't even know that anesthesia ends the life of such a fetus before it comes down the birth canal."

- *USA Today* said in an editorial opposing the bill (Nov. 3), "The fetus dies from an overdose of anesthesia given to its mother."

- *St. Louis Post-Dispatch* news story, Nov. 3: "The fetus usually dies from the anesthesia administered to the mother before the procedure begins."

SPREADING MYTH THAT ANESTHESIA KILLS, PG. 5

- Senator Carol Moseley-Braun (D-Il.) said during Senate floor debate on the bill (Nov 8), "The fetus dies during the first dose of anesthesia."

- Prior to the November 1 House vote on the bill, Planned Parenthood circulated to lawmakers a "fact sheet" titled, "H.R. 1833, Medical Questions and Answers," which includes this statement:

> "Q: When does the fetus die?
>
> "A: The fetus dies of an overdose of anesthesia given to the mother intravenously. A dose is calculated for the mother's weight which is 50 to 100 times the weight of the fetus. The mother gets the anesthesia for each insertion of the dilators, twice a day. This induces brain death in a fetus in a matter of minutes. Fetal demise therefore occurs at the beginning of the procedure while the fetus is still in the womb."

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Reply to: Washington, DC

January 27, 2012

Congressman Trent Franks
House of Representatives
2435 Rayburn House Office Building
Washington, D.C. 20515

Re: District of Columbia Pain-Capable Unborn Child Protection Act

Dear Congressman Franks:

Liberty Counsel Action, on behalf of more than 750,000 members nationwide, would like to express support for the District of Columbia Pain-Capable Unborn Child Protection Act. Liberty Counsel Action focuses on issues relating, in part, to the sanctity of human life, and this Act ensures both, expectant mothers and their unborn children, receive the best care available.

It is most certainly in the best interest of expectant mothers for doctors to perform thorough examinations of the mother and her unborn child prior to an abortion procedure. Information like the post-fertilization age of the unborn child assists the mother in making an informed decision about an abortion and assists the doctor in knowing how best to care for his patients.

Abortions are gruesome procedures that have lasting effects on women. Studies show abortions can have physiological impacts, such as an increased risk of breast cancer, and the psychological effects of consenting to the taking of a human life should not be underestimated. This bill will not only save lives by preventing abortions after twenty weeks gestation, but will hopefully, save the consciences of women who would otherwise endure the lifelong guilt of having subjected her unborn child to severe pain during the abortion process.

Thank you, Congressman Franks, for introducing the District of Columbia Pain-Capable Unborn Child Protection Act. We look forward to favorably scoring the bill when it makes it to the floor for a vote.

Sincerely,

Mandi Campbell
Director of Public Policy

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Consciousness without a cerebral cortex: A challenge for neuroscience and medicine

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Abstract: A broad range of evidence regarding the functional organization of the vertebrate brain – spanning from comparative neurology to experimental psychology and neurophysiology to clinical data – is reviewed for its bearing on conceptions of the neural organization of consciousness. A novel principle relating target selection, action selection, and motivation to one another, as a means to optimize integration for action in real time, is introduced. With its help, the principal macrosystems of the vertebrate brain can be seen to form a centralized functional design in which an upper brain stem system organized for conscious function performs a penultimate step in action control. This upper brain stem system retained a key role throughout the evolutionary process by which an expanding forebrain – culminating in the cerebral cortex of mammals – came to serve as a medium for the elaboration of conscious contents. This highly conserved upper brainstem system, which extends from the roof of the midbrain to the basal diencephalon, integrates the massively parallel and distributed information capacity of the cerebral hemispheres into the limited-capacity, sequential mode of operation required for coherent behavior. It maintains special connective relations with cortical territories implicated in attentional and conscious functions, but is not rendered nonfunctional in the absence of cortical input. This helps explain the purposive, goal-directed behavior exhibited by mammals after experimental decortication, as well as the evidence that children born without a cortex are conscious. Taken together these circumstances suggest that brainstem mechanisms are integral to the constitution of the conscious state, and that an adequate account of neural mechanisms of conscious function cannot be confined to the thalamocortical complex alone.

Keywords: action selection; anencephaly; central decision making; consciousness; control architectures; hydranencephaly; macrosystems; motivation; target selection; zona incerta

1. Introduction

The four semi-independent pacemakers of the non-cephalized nervous system of the cubomedusa equip this predatory jellyfish with flexible directional locomotor responsiveness to asymmetric sensory inputs (Satterlie & Nolen 2001). There is no reason to assume that the environmental guidance thus supplied by its radially arranged nerve net, involves or gives rise to experience of any kind. Our own environmental orientation, on the other hand, commonly takes place in a state of wakefulness we call conscious, which typically involves seeing, hearing, feeling, or other kinds of experience. Somewhere between medusa and human there is a transition to conscious function, and the nature of the capacity it bestows has exercised psychology, neuroscience, and cognitive studies virtually since their inceptions (Adrian et al. 1954; Baars 1988; James 1890/1983; Mandler 1975).

There is no compelling reason to think that nervous systems more complex than those of the medusa, and capable of performing more sophisticated functions, should not also perform in a perpetual night of unconsciousness. The fact that not all of them do so suggests that consciousness has some role or function to fill in the neural economy of brains thus endowed (Searle 1992). In exploring what this might involve, the exclusive concern throughout what follows will be with consciousness in its most basic and general sense, that is, as the state or condition presupposed by any experience whatsoever. Given recent proliferation of terminology surrounding the concept of consciousness (see Morin 2006 for a useful analysis and integration), the following additional remarks should help place this usage in context.

As employed here, the attribution of consciousness is not predicated upon any particular level or degree of complexity of the processes or contents that constitute the conscious state, but only upon whatever arrangement of those processes or contents makes experience itself possible. To the extent that any percept, simple or sophisticated, is experienced, it is conscious, and similarly for any feeling, even if vague, or any impulse to action, however inchoate.

BJORN MERKER is a neuroscientist with longstanding interest in brain mechanisms of consciousness: In an undergraduate term paper of 1971 he proposed the thalamic reticular nucleus as a central mechanism of attention on the basis of its anatomy and inhibitory connectivity. He obtained his doctorate from the Department of Psychology and Brain Science at M.I.T. in 1980 with a dissertation on the hamster colliculus. Since then he has worked on oculomotor physiology in cats, on the primary visual cortex in macaques, on song development and mirror self-recognition in gibbons, and on the evolutionary and developmental background to human music.

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This agrees well with the type of dictionary definition that renders consciousness as "the state or activity that is characterized by sensation, emotion, volition, or thought" (*Webster's Third New International Dictionary*, unabridged edition, 1961). In this basic sense, then, consciousness may be regarded most simply as the "medium" of any and all possible experience.

With regard to the way in which this medium might be implemented neurally, the present treatment is committed to an architectonic rather than a quantitative (or "graded") view. That is, as here conceived, a conscious mode of functioning is dependent upon quite specific neural arrangements creating interfaces of particular kinds between specific domains of neural function, rather than a result of a general increase in informational capacity or complexity achieved by expansion of a structural substrate which below a certain size does not support consciousness. Thus, what disqualifies the medusa nerve net in this regard is *not* its simplicity, but its lack of specific structural arrangements required to support conscious function. Given an arrangement capable of supporting consciousness, its contents may differ widely in complexity or sophistication. The range of possibilities in this regard is felicitously captured by the "scale of sentience" of Indian tradition (Bagchi 1975), as follows:

> "This."
> "This is so."
> "I am affected by this which is so."
> "So this is I who am affected by this which is so."

Each "stage" in this scale, from mere experienced sensation to self-consciousness, falls within the compass of consciousness as here defined, and presupposes it. Accordingly, to see, to hear, to feel, or otherwise to experience something is to be conscious, irrespective of whether in addition one is aware that one is seeing, hearing, and so forth, as cogently argued by Dretske (1995; see also Merker 1997; Searle 1992). Such additional awareness, in reflective consciousness or self-consciousness, is one of many contents of consciousness available to creatures with sophisticated cognitive capacities. However, as noted by Morin (2006), even in their case, it is present only intermittently, in a kind of time-sharing with more immediate, unreflective experience. To dwell in the latter is not to fall unconscious, but to be unselfconsciously conscious. Reflective awareness is thus more akin to a luxury of consciousness on the part of certain big-brained species, and not its defining property.

The exploration of the constitution of the conscious state to be pursued here will yield a conception of its functional role revolving around integration for action. As such, its functional utility will turn out to be independent of the level of sophistication at which the contents it integrates are defined. This opens the possibility that the evolution of its essential mechanisms did not have to await advanced stages of cortical development, but took place independently of it. As we shall see, certain fundamental features of vertebrate brain organization suggest that key mechanisms of consciousness are implemented in the midbrain and basal diencephalon, while the telencephalon serves as a medium for the increasingly sophisticated elaboration of conscious contents.

With some notable exceptions (e.g., Bogen 1995; Brown 1989; Panksepp 1982; Parvizi & Damasio 2001; Scheibel & Scheibel 1977; Sewards & Sewards 2000; Thompson 1993; Watt 2000), brainstem mechanisms have not figured prominently in the upsurge of interest in the nature and organization of consciousness that was ushered in with cognitivism in psychology and neuroscience (Baars 1988; Mandler 1975; Miller 1986). Few cognitivists or neuroscientists would today object to the assertion that "cortex is the organ of consciousness."[1] This is, in a sense, a return to an older view of the supremacy of the cerebral cortex from which a fundamental discovery of the late 1940s had stimulated a partial retreat. In keeping with the sense that the cerebral cortex is the organ of higher functions, it had been widely assumed that the regulation of its two primary states – sleep and wakefulness – was a cortical function, as well (see, e.g., the critical discussion of this stance in Gamper 1926, pp. 68–78). Then, in the late 1940s, Moruzzi and Magoun (1949) discovered that local stimulation of circumscribed cell groups in the pons and midbrain of experimental animals exerts a global activating influence on the cerebral cortex as well as on behavioral state, and that experimental lesions in these brainstem sites are capable of rendering animals somnolent and even comatose (Magoun 1954; cf. Parvizi & Damasio 2003). This came as a shock to the corticocentric perspective, and stimulated an avalanche of research on brainstem regulation of sleep and wakefulness and its relationship to the conscious state (summarized in symposium volumes edited by Adrian et al. 1954; Jasper et al. 1958; and Eccles 1966).

These efforts proved to be so successful that the once daring proposal that the brainstem regulates cortical state is unproblematic today. The same cannot be said of an allied, largely neglected, but even more radical proposal that emerged from the same pioneering wave of consciousness studies. Some of the principals in these developments – notably the neurosurgeon Wilder Penfield and his colleague Herbert Jasper – went on to re-examine the routine assumption that another "higher function," closely allied to that of sleep and wakefulness, namely consciousness, is an exclusively cortical affair (Penfield & Jasper 1954). On the basis of a set of clinical and physiological observations centered on the epilepsies, these authors proposed that *the highest integrative functions of the brain are not completed at the cortical level, but in an upper brainstem system of central convergence supplying the key mechanism of consciousness* (Penfield 1952). As their proposal is the natural point of departure for the present one, which elaborates and updates it in the light of subsequent developments, a brief review of its history follows.

2. Clinical beginnings

Penfield and Jasper left the anatomical definition of the upper brainstem system they invoked somewhat vague, but it was suggested to include the midbrain reticular formation and its extension into what was then known as the "nonspecific" thalamus (a nuclear grouping encompassing the midline, intralaminar, and reticular thalamic nuclei). They regarded this anatomically subcortical system to be functionally supra-cortical in the sense of occupying a superordinate position relative to the cerebral cortex in functional or control terms (Penfield & Jasper 1954,

pp. 28, 77; see sects. 3 and 4 of the target article following). They called it the "centrencephalic system," and assigned it a crucial role in the organization of conscious and volitional functions (ibid., p. 473). Figure 1 is based on a figure illustrating A. Fessard's lucid account of the conceptual setting for these ideas, included in the first of the symposium volumes cited earlier (Fessard 1954).

The Penfield and Jasper proposal emerged from extensive experience derived from an innovation in neurosurgical practice: they routinely removed sizeable sectors of cortex in conscious patients for the control of intractable epilepsy (Penfield & Jasper 1954). By performing the surgery under local anesthesia only, the authors ensured that their patients remained conscious, cooperative, and capable of self-report throughout the operation. This allowed the neurosurgeons to electrically stimulate the exposed cortex while communicating with the patient, in order to locate functionally critical areas to be spared when removing epileptogenic tissue. They then proceeded to remove cortical tissue while continuing to communicate with the patient. They were impressed by the fact that the removal of sizeable sectors of cortex such as those diagrammed in the composite of Figure 2 never interrupted the patient's continuity of consciousness even while the tissue was being surgically removed.

Penfield and Jasper note that a cortical removal even as radical as hemispherectomy does not deprive a patient of consciousness, but rather of certain forms of information, discriminative capacities, or abilities, but not of consciousness itself (Penfield & Jasper 1954, p. 477; cf. Devlin et al. 2003). That does not mean that no cortical insult is capable of compromising consciousness. In adult humans massive

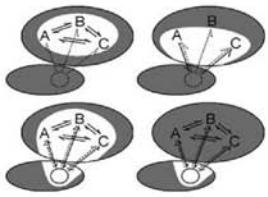

Figure 1. Four principal alternatives regarding interactions between cortex and brainstem in the constitution of the conscious state. Cortex (large oval) and brainstem (small oval) in highly schematic side (saggittal) view. Small circle: "centrencephalic system." In each alternative, normal waking cortical function is assumed to require "enabling" activation originating in the brain stem, marked by three dashed arrows radiating from brainstem to cortex. **Upper left**: the "corticocentric" alternative, in which integration through cortico-cortical connections alone is sufficient to constitute the conscious state. **Upper right**: Cortical integration via a subcortical relay, such as might occur via the dorsal thalamus. Only one such relay is depicted for the sake of clarity. The scheme is still corticocentric, since integration is cortical, albeit dependent upon extracortical relays for its implementation. **Lower left**: Centrencephalic hypothesis, based on diagram IV in Fessard (1954). Here an essential functional component of consciousness is supplied by brainstem mechanisms interacting with the cortex. **Lower right**: Primary consciousness implemented in the brainstem alone, as in cases of cortical removal or damage discussed in sections 4.4 and 5 of the text.

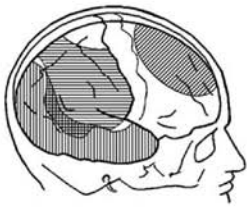

Figure 2. Large cortical excisions performed under local anesthesia by W. Penfield for the control of intractable epilepsy in three patients, entered on a single diagram. The patients remained conscious and communicative throughout the operation. All removals extended to the midline. The two posterior cases were right-sided, whereas the frontal removal was left-sided, and has been mirror-imaged. In no case was the removal of cortical tissue accompanied by a loss of consciousness, *even as it took place*. (Redrawn after figures VI-2, XIII-2, and XVIII-7 of Penfield & Jasper 1954.)

bilateral cortical damage will typically issue in a so-called persistent vegetative state (Jennett 2002). This by itself does not, however, allow us to make an equation between cortical function and consciousness, because such damage inevitably disrupts numerous brainstem mechanisms normally in receipt of cortical input, as discussed further in subsequent sections (see Shewmon 2004 for the conceptual and empirical complexities of the vegetative state). What impressed Penfield and Jasper was the extent to which the cerebral cortex could be subjected to acute insult without producing so much as an interruption in the continuity of consciousness. Their opinion in this regard bears some weight, in that their magnum opus of 1954 – *Epilepsy and the Functional Anatomy of the Human Brain* – summarizes and evaluates experience with 750 such operations.

When the exposed cortex was stimulated electrically to assess functional localization, stimulation parameters were adjusted so as to avoid triggering epileptic seizures in the patient. From time to time seizures were nevertheless triggered inadvertently. Over the large number of operations performed, every variety of seizure was thus produced by cortical stimulation, except one: Penfield and Jasper never saw the complete electrographic pattern that accompanies absence epilepsy induced by electrical stimulation of any part of the cerebral cortex (Penfield & Jasper 1954, p. 480). This pattern of 3 per second trains of "spike and wave" discharges evolves synchronously in the two hemispheres, down to a coincidence in the two hemispheres of the very first abnormal spike detectable in the electroencephalogram (Gibbs et al. 1936, 1937; Penfield & Jasper 1954, p. 483, Fig. XII-3, p. 624, Fig. XV-26, etc.).

Seizures of this type bear directly on our topic because of their conspicuous association with disturbances of consciousness (Penfield & Jasper 1954, pp. 24, 28). In fact, they are often *initiated* by a lapse of consciousness (p. 477), and in pure form they "consist almost solely of a lapse of consciousness" (p. 480). Without a preceding "aura" or other warning, and in the midst of normal activities, the patient assumes a vacant expression ("blank

stare") and becomes unresponsive. Ongoing activities may continue in the form of automatisms (as complex as automatic speech, implying organized cortical activity), or they may arrest for the duration of the often-brief seizure episode. At the end of such a seizure, which may last no more than a few seconds, the patient, who typically remains upright throughout, sometimes actively moving, resumes conscious activities where they were interrupted, has amnesia for what transpired during the episode, and may have no knowledge that the episode took place except indirectly, by means of evidence for the lapse of time available to the discursive, post-seizure, intellect.

Penfield and Jasper recognized in these seizures "a unique opportunity to study the neuronal substratum of consciousness" (Penfield & Jasper 1954, p. 480; cf. Blumenfeld & Taylor 2003). The coincident bilateral onset and cessation of these seizures suggested to the authors an origin in a centrally placed upper brainstem site of paroxysmal induction (Penfield & Jasper 1954, pp. 27, 473, 477, 482, 622–633). Though in their experience the pattern was not triggered by cortical stimulation, it could be evoked experimentally in the cat by stimulation of the midline thalamus (Jasper & Droogleever-Fortuyn 1947). Modern methods have added both detail and qualifications to the Penfield and Jasper account (see review by Meeren et al. 2005), yet upper brainstem involvement in absence epilepsy has stood the test of time, and is still being actively pursued both clinically and through research employing animal models (Blumenfeld & Taylor 2003; Danober et al. 1998; Derensart et al. 2001; McCormick & Contreras 2001; Stefan & Snead 1997; Strafstrom 2006). We shall return to this matter in Section 4.5.3.

Penfield and Jasper stressed that the postulated centrencephalic system is *symmetrically related to both cerebral hemispheres* (in the sense of radial rather than bilateral symmetry (see Penfield & Jasper 1954, p. 43, and figures on pp. 145 and 173). They denied that this system "functions by itself alone, independent of the cortex" and suggested instead that it "functions normally only by means of employment of various cortical areas" (Penfield & Jasper 1954, pp. 473–474). They conceived of it as a convergently innervated upper brainstem system serving to coordinate and integrate the functional economy of the forebrain as a whole, intimately involved in conscious and volitional functions, as well as in the laying down of memories across the lifespan (Penfield & Jasper 1954, pp. 140–145, 282).

3. Bringing the centrencephalic proposal up to date

A valuable review of the centrencephalic proposal, in light of developments up till the end of the 1980s, is provided by Thompson (1993, published posthumously). He calls attention to the relevance of the clinical literature on so called "subcortical dementia" to the centrencephalic theory, and further suggests that animal evidence for a subcortical "general learning system" may supply some of the anatomical detail left unspecified by Penfield and Jasper. This "general learning system" is defined by neural structures which, when damaged, produce deficits in *each* member of a set of highly diverse learning tests for rats. As identified through a long-term research program conducted by Thompson and colleagues, it consists of the basal ganglia, including the *substantia nigra* and ventral tegmental area, ventrolateral thalamus, superior colliculus, median raphé, and pontine reticular formation. The functional significance of key members of this constellation (which has access to sensory information independently of the cortex) is considered in some detail in Section 4 of the target article, for which the following preliminary considerations will set the stage.

The central claim of the Penfield and Jasper hypothesis is a claim regarding systems-level organization of neural functions. The idea that a system can be "anatomically subcortical but functionally supra-cortical" is a statement about brain macrosystems and how they relate and interact with one another. It is most easily approached from the side of the "final common path" of all brain output as far as actual behavior is concerned, namely brainstem and spinal motoneuron pools. Not only are these clusters of final output cells invariably innervated by multiple sources of afference (Graf et al. 2002; Kuypers & Martin 1982; Nudo & Masterton 1988; Ugolini 1995), but individual motoneurons receive synaptic input from diverse sources utilizing different transmitters (Holstege 1991; Wentzel et al. 1995). These sources include spinal and brainstem pattern generators (Grillner 2003), various territories of the brain stem reticular formation (Jordan 1998), and a multitude of both direct and indirect brainstem and forebrain afferents, among which the indirect ones often are relayed via the reticular formation (Zahm 2006).

Thus, the fact that the motor cortex maintains direct connections with brainstem and spinal motoneurons by no means implies that it ever is in sole command of behavior. At every level of its descending innervation of motoneuron pools it is only one of many inputs determining final outcomes. Moreover, the motor cortex accounts for just a fraction of descending cortical output, and is responsible for only select forms of distal behavior (Lang & Schieber 2003; Lawrence & Kuypers 1968; Kuypers 1982, 1987). In such a setting, the idea that the output of a subcortical structure might override a cortical one, and in this sense could exercise supra-cortical control over behavior, is hardly controversial. When an act of deliberate effort (say driven by prefrontal executive systems) is successful in overriding or inhibiting a given behavioral tendency, the cortex is in command of behavior, temporarily exercising determining control over its course. The fact that such effort does not always succeed (say in the face of sufficient magnitudes of fear, hunger, or pain) means that the frontal executive can be overridden by more primitive mechanisms. When a subcortical source prevails in such competitive interactions, an anatomically subcortical system has exercised supra-cortical functional control over behavior.

It is necessary, in other words, to distinguish "higher" in the sense of cognitive sophistication from "higher" in control terms. In this light, the Penfield and Jasper proposal amounts to a claim that certain upper brainstem systems in receipt of convergent cortical projections occupy a superordinate position in the latter sense. As I detail further in subsequent sections, the diverse hemispheric as well as brainstem input to these structures equips them for the kind of superordinate decision

making crucial for the global sequencing and control of behavior (Prescott et al. 1999). It is also within processes dedicated to "integration for action" that we can find a well-defined functional role for a particular mode of neural organization that qualifies as conscious, in good agreement with the Penfield and Jasper proposal. To set the stage for a treatment of that more demanding topic in sections 4 and 5, two lines of evidence regarding brainstem function that bear on their proposal are briefly reviewed.

3.1. The Sprague effect

Complete removal of the posterior visual areas of one hemisphere in the cat (parietal areas included) renders the animal profoundly and permanently unresponsive to visual stimuli in the half of space opposite the cortical removal (Sprague 1966; see also, Sherman 1974; Wallace et al. 1989). The animal appears blind in a manner resembling the cortical blindness that follows radical damage to the geniculostriate system in humans. Yet inflicting *additional* damage on such a severely impaired animal at the midbrain level *restores* the animal's ability to orient to and to localize stimuli in the formerly blind field (Sprague 1966; cf. Sherman 1977; Wallace et al. 1989). This is accomplished by removing the contralateral superior colliculus or by an intervention as small as a knife-cut that severs fibers running in the central portion of the collicular commissure. That is, adding a small amount of damage in the brainstem to the cortical damage "cures" what appeared to be a behavioral effect of massive cortical damage. The restored visual capacity is limited essentially to the ability to orient to and approach the location of moving visual stimuli in space (Wallace et al. 1989). Visual *pattern* discrimination capacity does not recover after the midbrain intervention (Loop & Sherman 1977), though the midbrain mechanism can be shown to play a role even in such tasks (Sprague 1991).

The Sprague effect is a consequence of secondary effects generated at the brainstem level by the unilateral cortical removal (Hikosaka & Wurtz 1989; Hovda & Villablanca 1990; Jiang et al. 2003). The damage not only deprives the ipsilateral superior colliculus of its normal and profuse cortical input (Berson & McIlwain 1983; Harting et al. 1992; Palmer et al. 1972; Sprague 1975), but it unbalances collicular function via indirect projection pathways. Chief of these is the powerful inhibitory projection from the substantia nigra to the colliculus, which crosses the midline in a narrow central portion of the collicular commissure (McHaffie et al. 1993; Sprague 1996; Wallace et al. 1990; for additional possibilities, see Durmer & Rosenquist 2001). The "restorative" interventions partially correct this imbalance, allowing the collicular mechanism to resume at least part of its normal functional contribution to behavior, with partial restoration of visually guided behavior as a result.

The point is underscored by the analogous circumstances pertaining to the neglect of one half of space (unilateral neglect) that follows more limited inactivation of the cortex (by reversible cooling) at the junction of occipital, parietal, and temporal lobes in one hemisphere of the cat. This neglect also lifts upon inactivation (by reversible cooling) of the superior colliculus opposite to the cortical inactivation (Lomber & Payne 1996). Analogous restorative effects of midbrain damage on neglect caused by frontal cortical damage have been observed in a human patient (Weddell 2004). Though the unawareness featured in cases of unilateral neglect in humans is far from a simple entity (see review by Mesulam 1999), it bears on our topic by being perhaps the closest approximation to an impairment that includes specific effects on consciousness produced by localized cortical damage (Driver & Vuilleumier 2001; Rees 2001; see also Jiang et al. 2003).

The Sprague effect demonstrates that hidden in the hemianopia or neglect caused by cortical damage lies a deficit on the part of a brainstem visual mechanism disabled as a secondary effect of the cortical removal. This means that a functional deficit following damage limited to the cortex cannot, as a matter of course, be taken to reflect an exclusively cortical contribution to functional capacity, because the deficit may reflect "remote" effects on brainstem systems, as well. As Sprague originally expressed it:

> The hemianopia that follows unilateral removal of the cortex that mediates visual behavior cannot be explained simply in classical terms of interruption of the visual radiations that serve cortical function. Explanation of the deficit requires a broader point of view, namely, that visual attention and perception are mediated at both forebrain and midbrain levels, which interact in their control of visually guided behavior. (Sprague 1966, p. 1547)

That conclusion agrees well with the Penfield and Jasper perspective reviewed in the foregoing; and it tells us that without cognizance of potential subcortical contributions to a deficit caused by cortical damage, the scope of functions attributed to the cortex will be counterfactually inflated.

3.2. Target selection in the midbrain

Although superficially inconspicuous, the superior colliculus in the roof ("tectum") of the midbrain exhibits considerable structural and functional complexity. Long known to play a role in "visual grasping" or "foveation" (Hess et al. 1946; Schiller & Koerner 1971), further study has revealed unexpected sophistication in its functional organization (Keller et al. 2005; Krauzlis et al. 2004; May 2005; Sparks 1999). It is the only site in the brain in which the spatial senses are topographically superposed in laminar fashion within a common, premotor, framework for multi-effector control of orienting (Merker 1980). Its functional role appears to center on convergent integration of diverse sources of information bearing on spatially triggered replacement of one behavioral target by another, and evidence is accumulating for a collicular role in target selection (Basso & Wurtz 1998, 2002; Carello & Krauzlis 2004; Cavanaugh & Wurtz 2004; Fecteau & Munoz 2006; Glimcher & Sparks 1992; Horowitz & Newsome 1999; Krauzlis et al. 2004; McPeek & Keller 2004; Schlag-Rey et al. 1992; Wurtz & Mohler 1974; see also Grobstein 1988, pp. 44–45). Such a role has direct implications for the topic of superordinate control functions.

A collicular role in target selection is unlikely to be a passive reflection of decisions taken in other structures. It is not fully accounted for by the powerful input it receives from the substantia nigra (Basso & Wurtz

2002), and the diversity of collicular afferents precludes any one of them from exercising sole control over collicular function. These afferents include a wide range of brainstem (Edwards 1980; Edwards et al. 1979) and visual as well as nonvisual cortical sources (Collins et al. 2005; Harting et al. 1992; 1997; Kawamura & Konno 1979; Sherman et al. 1979). Cortical afferents are monosynaptic, originating in layer V pyramidal cells, placing the colliculus as close to the cortex as two cortical layers are to one another. In the cat they include some 17 visual areas (Harting et al. 1992), and in primates there are contributions from both the dorsal (parietal cortex) and the ventral (temporal cortex) "streams" of the visual system (Fries 1984; Steele & Weller 1993; Webster et al. 1993). Any sensory modality used in phasic orienting behavior appears to receive obligatory representation in the colliculus. Besides the major spatial senses of vision, audition, and somesthesis, they include pain (Wang & Redgrave 1997) and exotic senses such as infrared (Hartline et al. 1978), electroceptive (Bastian 1982), magnetic (Nemec et al. 2001), and echolocation systems (Valentine & Moss 1997), depending on species.

In the colliculus these diverse convergent inputs are arranged in topographically organized sheets layered one upon the other through the depths of the colliculus (Harting et al. 1992; May 2005). Intrinsic collicular circuitry distributes excitatory as well as inhibitory collicular activity within and across layers and across major collicular subdivisions (Behan & Kime 1996; Bell et al. 2003; Binns 1999; Doubell et al. 2003; Lee et al. 1997; Meredith & King 2004; Meredith & Ramoa 1998; Mize et al. 1994; Özen et al. 2000; Zhu & Lo 2000). There is therefore no dirth of complex intrinsic collicular circuitry – only beginning to be systematically charted – for collicular decision-making based upon its diverse sources of afference.

The collicular role in target selection is accordingly likely to be causal (Carello & Krauzlis 2004; McPeek & Keller 2004; see also Findlay & Walker 1999; Yarrow et al. 2004; and sect. 4.2 of the target article). This would place the colliculus at the functional top rather than bottom of control processes in its domain. The selection of a target for behavior is the brain's final output in that regard. It is the pivotal event for which all other processes are but a preparation, summing them up in the actual decision to settle on one target for action rather than another (Allport 1987; Brooks 1994; Dean & Redgrave 1984; Isa & Kobayashi 2004; McFarland & Sibly 1975; Tyrrell 1993; see Feetcau & Munoz 2006 for collicular "priority mapping" in relation to action).

The functional prediction from the loss of such a structure is not the absence of target acquisition, but its impoverishment. Not only is the brain redundantly organized in this regard (Lomber et al. 2001; Schall 1997; Schiller et al. 1979; Tehovnik et al. 1994), but the loss of a superordinate function in a layered control architecture does not disable the system as a whole (Brooks 1986, 1989; Prescott et al. 1999), just as a well organized army need not cease functioning on the loss of its commander. A macaque with experimental collicular lesions is not incapable of moving its eyes onto targets, but exhibits a reduced variety of eye and orienting movements and is indistractible, a common finding in other species as well (Albano & Wurtz 1978; Casagrande & Diamond 1974; Denny-Brown 1962; Goodale & Murison 1975; Merker 1980; Mort et al. 1980; Schiller et al. 1979; Schiller & Lee 1994; Schneider 1967). This may reflect a compromised *scope* and *sophistication* of target selection, and the role of the intact colliculus would accordingly instantiate the Penfield and Jasper conception of a *highest integrative function* which, while *anatomically subcortical*, is *functionally supra-cortical*.

4. Integration for action

As noted in section 3, in drawing the contrast between "higher" in cognitive terms and "higher" in control terms, competition for control over behavior ends only at the stage of the "final common path" of motoneurones. It is along that approach, among upper brainstem mechanisms of "integration for action," that we shall identify a prototype organization for conscious function. The issue takes us to the very origin of the vertebrate brain plan, which is not only cephalized, but centralized. Not all animals rely on centralized neural organization to control behavior, even when possessed of a brain. A number of invertebrate forms, including insects, concentrate considerable neural resources to segmental ganglia. Their brain is in a sense no more than the anterior-most of these ganglia, in receipt of the output of the specialized receptors of the head. It does not necessarily exercise a command function in the sense of central control of behavior (see Altman & Kien 1989).

The decentralized neural control of an insect such as the ant allows its body to survive without its brain. Moreover, if given adequate somatic stimulation in this condition, it will perform many of the complex behaviors in its repertoire with apparent competence, though naturally without relation to the distal environment (Snodgrass 1935). A vertebrate, on the other hand, does not survive for more than seconds after the loss of its brain, because in vertebrates even vital functions are under central brain control. The difference with respect to insects is underscored by the contrasting disposition of motor neurons. In insects, they are concentrated to segmental ganglia but are rare in the brain (Snodgrass 1935), whereas in vertebrates they populate the brain in sets of distinctively organized motor nuclei. Motor control in vertebrates has "moved up," as it were, to that end of the neuraxis which leads in locomotion and is in receipt of the output of the chief exteroceptors (cf. Grillner et al. 1997).

The basic organizational features of the vertebrate brain are highly conserved across taxa despite unequal development of one or another of its senses or subdivisions (Nieuwenhuis et al. 1998). All vertebrates, that is, have "in outline" the same brain plan, assembled from primitive beginnings in chordate ancestry (Butler & Hodos 1996; Holland & Holland 1999; Northcutt 1996b). The prominent role of large, image-forming eyes and their central connections in this development came to exert a profound effect on the manner in which the vertebrate brain plan was centralized, with implications for our understanding of the way in which "higher" in cognitive terms relates to "higher" in control terms. That development involves the integrative machinery straddling the so-called synencephalon, or junction between midbrain and diencephalon – to which we now turn.

4.1. The synencephalic bottleneck and how the vertebrate brain came to be centralized around it

There was a time in prevertebrate ancestry when the midbrain and diencephalon alone, or rather the first rostral differentiations of the neural tube that can be homologized with the vertebrate midbrain and diencephalon, constituted the functionally highest and also anatomically most rostral subdivision of the neuraxis (Holland & Holland 1999, 2001; Holland et al. 1994; Lacalli 1996, 2001; Wicht 1996). It housed the neural circuitry connecting a primitive, unpaired "frontal eye" and other rostral sensory equipment (Lacalli 1996) with premotor cells in cephalochordate filter feeders (represented today by *Amphioxus*, the lancelet). As far as is known, cephalochordate filter feeders lacked a sense of smell, and they were without a telencephalon altogether (Butler 2000; Holland et al. 1994).

Though our brain nomenclature historically groups the diencephalon together with the telencephalon to make up the forebrain, there is nothing fundamental about such a grouping, as the just mentioned phylogenetic circumstances show. Rather, for what follows it will be convenient to retain the primitive grouping of midbrain and diencephalon together under the label mesodiencephalon or "optic brain." In all vertebrates these two segments of the neuraxis, along with the transitional "synencephalon" (pretectum) wedged between them, house the primary terminations of the optic tract (cf. Butler 2000). The latter covers their external surfaces in the form of a ribbon of fibers running obliquely from the optic chiasm beneath the hypothalamus across the diencephalon and mesencephalon up to the latter's roof ("tectum"). Along the way it innervates structures as different as the hypothalamus, ventral thalamus, dorsal thalamus, pretectum, accessory optic nuclei, and superior colliculus (tectum). The same territory also houses some of the major *integrative structures of broad functional scope common to all vertebrates* (see Fig. 3).

The principal poles of this integrative machinery are the hypothalamus forming the floor of the diencephalon, on the one hand, and the superior colliculus forming the roof of the midbrain, on the other. The former is an intricate nuclear aggregate critical for the mutual regulation and integration of a vertebrate's entire repertoire of goal-directed, motivated behavior covering exploratory, foraging, ingestive, defensive, aggressive, sexual, social, and parental modes of behavior (Swanson 2000), to name the principal ones. The other pole, colliculus/tectum, serves the intermodal integration of the spatial senses by which vertebrates relate to their surroundings via coordinated orienting movements of eyes, head, and body, as already summarized in section 3.2. Between these two is wedged additional integrative machinery in the form of the midbrain reticular formation, ventral thalamus, the periaqueductal gray, the ventral tegmental/substantia nigra pivot of the striatal system, as well as "locomotor centers" and basic mechanisms serving navigation. I will return to some of these in subsequent sections.

This concentration of conserved integrative machinery to the mesodiencephalon, I suggest, reflects the costs and benefits of evolving image-forming eyes in the ancestors of vertebrates (cf. Northcutt 1996a). Full use of the potential powers of visual guidance meant evolving

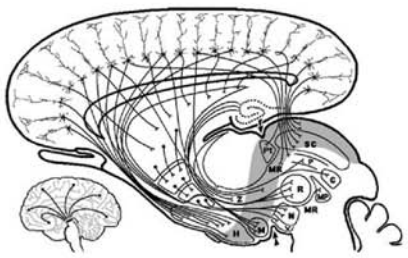

Figure 3. Schematic saggittal diagram depicting cortical convergence (in part via the basal ganglia) onto key structures in the region of the "synencephalic bottleneck" (marked by thick arrows in the main figure and by a black bar in the inset). Abbreviations: **C**, nucleus cuneiformis; **H**, hypothalamus (preoptic area included); **M**, mammillary bodies; **MP**, "mesopontine state control nuclei" (locus coeruleus, pedunculopontine and laterodorsal tegmental nuclei, and dorsal raphé); **MR**, midbrain reticular formation; **N**, substanta nigra; **P**, periaqueductal gray matter; **Pt**, pretectum; **R**, red nucleus; **SC**, superior colliculus; **V**, ventral tegmental area; **Z**, zona incerta. The dual axon seen issuing from some of the pyramidal cells of cortical layer 5 is an illustrative convenience only. Shaded region marks the surface course of the optic tract.

solutions to an intricate set of sensorimotor problems. The confounding of sensory information by the sensory consequences of movement ("re-afference"; von Holst & Mittelstaedt 1950) is particularly problematic for image-forming eyes, requiring their stabilization with respect to the world during movement. This is done by vestibular counter-rotation punctuated by quick resets of the eyes, which concentrates blurring-time to the brief reset episodes. Thus, *vision alone among all the senses features independent spatial mobility of the receptor array itself*, and a full-fledged oculomotor system evolved in the immediate ancestors of true vertebrates (Braun 1996, p. 272; Fritsch et al. 1990; Wicht 1996, p. 253). The reflex circuitry connecting vestibular and oculomotor nuclei, centered on the medial longitudinal fasciculus, is also among the most conservative and basic features of the brainstem in all vertebrates (Carpenter 1991; Windle & Baxter 1936).

Yet, with eyes free to turn in their orbits, there is no longer a fixed relation between retinal location and spatial direction relative to body or head, nor to the localizing function of any sensory modality which (in whole or in part) bears a fixed relation to the head. Hence the need for intermodal integration, for which the sensory integrating mechanism of colliculus/tectum – present in the roof of the midbrain of even jawless vertebrates – provides the basic, early and conserved solution (Iwahori et al. 1999; Zompa & Dubuc 1996). But once these basic problems of vision were solved, a bonus was within reach: Mobile eyes present a highly efficient means for sampling the environment, provided their control can be linked to motivational mechanisms ensuring their appropriate deployment in accordance with shifting needs.

It appears, in other words, that as the vertebrate brain plan took shape in prevertebrate ancestry under pressure of the evolution of mobile, image-forming eyes, a central

association between optic control circuitry and major neural mechanism for the integration of behavior/action were forged in segments of the neuraxis covered and innervated by the optic tract (cf. Fig. 3). At the time when this optic orienting machinery and associated integrative mechanisms evolved, the forebrain was still dominated by olfaction (Braun 1996; Northcutt & Wicht 1997; Wicht & Northcutt 1992). The sense of smell added no fundamentally new control requirements comparable to those of vision, and olfaction accordingly could be integrated with the mesodiencephalic control system by caudally directed fiber projections. These simply happen to arrive at the "optic brain" from an anterior direction, whereas other sensory afferents reach it from a caudal direction (somatosensory, octavolateral, i.e., vestibular/auditory/lateral line/electrosensory, etc.), or directly "from the side" through the optic tract (cf. Butler 2000).

Indeed, however much the telencephalon subsequently expanded, even to the point of burying the mesodiencephalon under a mushrooming mammalian neocortex, no other arrangement was ever needed, and that for the most fundamental of reasons. No efferent nerve has its motor nucleus situated above the level of the midbrain. This means that the very narrow cross-section of the brainstem at the junction between midbrain and diencephalon (synencephalon, marked by arrows in the main part of Fig. 3 and by a black bar in the inset) carries the total extent of information by which the forebrain is ever able to generate, control, or influence behavior of any kind. If, therefore, integration is for action, as proposed here for the mesodiencephalic control system, information-theory poses no obstacle to having an expansive neocortex make its contribution in this regard by convergent projections onto the highly conserved and pre-existing machinery of the midbrain and basal diencephalon, which therefore could retain its old integrative functions (see Fig. 3). Indeed, a bottleneck of this kind is exactly what is needed in order to convert the massively parallel and distributed information capacity of the cerebral hemispheres into a limited-capacity, sequential mode of operation featured in action selection for coherent behavior (Allport 1987; Baars 1993; Cabanac 1996; Cowan 2001; Mandler 1975; 2002, Ch. 2; McFarland & Sibly 1975; Tyrrell 1993).

That is, one need not know anything more about the vertebrate brain than the fact that its most rostral motoneurons are located *below* the synencephalic bottleneck, to know that the total informational content of the forebrain must undergo massive reduction in the course of its real-time translation into behavior. In the setting of such obligatory "data reduction" in a stretch of the neuraxis hosting major systems for the global regulation of behavior, a so far unrecognized optimizing principle lies hidden in the mutual dependency that links the motivational, the sensory, and the action selection requirements of the brain's control tasks. They form a "selection triangle," the principle of which is introduced here for the first time. The efficient neural implementation of this principle may harbor the secret of conscious function itself.

4.2. The "selection triangle": A proposed key to conscious function

Elementary necessities of animal existence such as food, shelter, or mates are not typically found in the same place at any given time, and they each require different and often incompatible behaviors. An animal's activities accordingly unfold under constraint of multiple goals or motives derived from the evolved and acquired needs it must fill through the sequence of its diverse actions over time (Baerends 1976; Tinbergen 1951). The tasks set by these goals compete for an animal's behavioral resources, and because the actions by which they are implemented are always confined to the present (where they typically are executed one at a time), their scheduling (action selection) features perpetual trade-offs in the time and effort that is allocated to them (McFarland & Sibly 1975). The ethological insight, that animal behavior rests upon a foundation of diverse goal functions that sometimes entail incompatible tasks or behaviors requiring sequencing/selection, entered the so-called behavior-based approach to robotics under the name "action selection" (Blumberg 1994; Brooks 1986; McFarland & Houston, 1981; Maes 1990; Prescott et al. 1999; Tyrell 1993; see also Meyer & Wilson 1991).

The needs reflected in the time budget of an animal's task allocations are, however, only one side of the equation of efficient decision-making. The fulfillment of needs is contingent on available *opportunities*. These are scattered in the world as ever-shifting targets of approach and avoidance among lively and often unpredictable contingencies within which they must be detected, located, and identified, often among multiple competing alternatives, all in real time. Interposed between the needs and their fulfillment through action on the world is the body with its appendages and other resources for getting about in the world and manipulating its objects. In concrete terms an action is a time series of bodily locations and conformations. These are what connect needs with opportunities. In so doing they themselves become a factor in singling out a given opportunity (target) for action (target selection). This is so because determining which one of several available potential targets is the best current choice for action will often depend, not on current needs alone, but additionally on the disposition of the body relative to those targets (in terms of its posture and position, movement trajectory, energy reserves, etc.; cf. Körding & Wolpert 2006).

In principle, each of the decision domains just invoked – action selection, target selection, and motivational ranking – may be defined in its own terms, without regard to the others. They may even make their contributions to behavior independently of one another (Altman & Kien 1989; Brooks 1986). But from the inherent functional relationship just sketched, that is, the fact that in terms of optimal performance *target selection is not independent of action selection, and neither of these is independent of motivational state* (reflecting changing needs), it follows that savings are achievable by exploiting that triangular dependency. It is not possible to reap the benefits of those savings short of finding some way of interfacing the three state spaces – each multidimensional in its own right – within some common coordinate space (decision framework) allowing their separate momentary states to interact with and constrain one another. This extends to such a tripartite interaction the principle already derived for the efficient management of motivational trade-offs, namely, the convertibility of different motives through a motivational "common currency"

and their convergence among themselves at some point in the system (McFarland & Sibly 1975; see also Cabanac 1992, and further in the present article).

The principle of a centralized brain system dedicated to this decision domain follows from this, though not the particulars of the three-way interface that must form its centerpiece. Evolving such an interface is far from a trivial problem, all the more so since its decisions must be made in real time. The brain, of course, has no direct access to either the target states of the world or the action states of the body that must be compared and matched in the light of motivational priorities. It is saddled with an inverse problem on both sensory and motor sides of its operations (Gallistel 1999; Kawato et al. 1993). The indirect reflections of relevant parameters to which it does have access, come to it, moreover, in diverse data formats. The differences between the spatial senses among themselves in this regard are mild compared to those between any one of these senses and the various musculoskeletal articulations and configurations they serve to control. How then might the former be compared with the latter? Add to this the already mentioned circumstance that every movement confounds the sensory information needed to guide behavior, and that the needs to be taken into account differ not only in urgency, but in kind, and the size of the design problem begins to emerge in outline.

To exploit the savings hidden in the functional interdependence between target selection, action selection, and motivation, this confounded complexity must be radically recast, to allow the three domains to interact directly in real time for the determination of "what to do next." It is the principal claim of the present target article that the vertebrate brain incorporates a solution to this decision problem, that it takes the general form of a neural *analog reality simulation* of the problem space of the tripartite interaction, and that the way this simulation is structured constitutes a conscious mode of function. It equips its bearers with veridical experience of an external world and their own tangible body maneuvering within it under the influence of feelings reflecting momentary needs, that is, what we normally call reality.[2] To this end it features an analog (spatial) mobile "body" (action domain) embedded within a movement-stabilized analog (spatial) "world" (target domain) via a shared spatial coordinate system, subject to bias from motivational variables, and supplying a premotor output for the control of the full species-specific orienting reflex. The crucial separation of body and world on which this arrangement hinges has recently been worked out in formal terms by David Philipona and colleagues (Philipona et al. 2003; 2004).

We have already seen in sections 3.2 and 4.1 that the roof of the midbrain of vertebrates houses a sophisticated laminar superposition of the spatial senses in a premotor framework for orienting. It appears to contain the essential signals for bringing these senses into registry (Groh & Sparks 1996; Jay & Sparks 1987; Krauzlis 2001; Populin & Yin 1998; Van Opstal et al. 1995; Zella et al. 2001) and for stabilizing the world relative to the body. Such stabilization is likely to utilize not only vestibular information (Bisti et al. 1972; Horowitz et al. 2005), but cerebellar "decorrelation" as well (Dean et al. 2002; 2004; cf. Guillaume & Pélisson 2001; Hirai et al. 1982; May et al. 1990; Niemi-Junkola & Westby 2000). The layered spatial maps in the roof of the midbrain would, in other words, represent the vertebrate brain's first bid for an analog simulation of a distal "world" (Scheibel & Scheibel 1977). We also saw that the other pole of the "optic brain," the hypothalamus, houses the basic circuitry for regulating and integrating motivational states related to goal-directed behaviors. Its output is brought to bear on the intermediate and deep layers of the superior colliculus not only by direct projections (Beitz 1982; Rieck et al. 1986), but indirectly, via massive and organized projections from hypothalamic nuclei to different sectors of the periaqueductal gray substance (Coto et al. 2005; see refs. 36, 37, 39, 222, & 256 in Swanson 2000).

The periaqueductal gray is a midbrain territory intimately related to the deeper collicular layers. It surrounds the cerebral aqueduct, and plays a critical role in the expression of a variety of emotion-related behaviors such as defensive, aggressive, sexual, vocal, and pain-related ones (Adams 1979; Behbehani 1995; Fernandez de Molina & Hunsperger 1962; Holstege et al. 1996; Jurgens 1994; Kittelberger et al. 2006; Lonstein et al. 1998; Mouton 1999; Panksepp 1982; 1998a; Watt 2000). Its longitudinal columns are functionally organized in terms of high-level tasks, goals, strategies, or contexts, such as "inescapable versus escapable pain" (Keay & Bandler 2002). It achieves particular prominence in mammals, and stimulating it electrically in conscious humans evokes powerful emotional reactions (Heath 1975; Iacono & Nashold 1982; Nashold et al. 1969). Functionally the periaqueductal gray is continuous and reciprocally interconnected with the immediately overlying deep layers of the superior colliculus (Bittencourt et al. 2005; Cadusseau & Roger 1985; Gordon et al. 2002; Grofova et al. 1978; Harting et al. 1992, Fig. 27; Sprague et al. 1961; Wiberg 1992). Here, then, in the intermediate and deep collicular connections with hypothalamus and periaqueductal gray, lies a connective interface between the brain's basic motivational systems and the orienting machinery of the collicular analog "world."

The third member of the selection triangle enters this system through the prominent projections from the substantia nigra to the intermediate collicular layers (Jiang et al. 2003; Mana & Chevalier 2001; see also sects. 3.1 & 3.2). Here the final distillate of basal ganglia action-related information is interdigitated with the latticework of histochemically defined compartements that organize the input-output relations of the intermediate colliculus (Graybiel 1978; Harting et al. 1997; Illing 1992; Illing & Graybiel 1986). It appears, in other words, that the territory extending from the dorsal surface of the midbrain to the aqueduct houses the connectivity needed to implement a three-way interface of the kind outlined in the foregoing discussion, and it is hereby proposed to do so. The elements of this scheme are sketched in Figure 4.

Such a conception fits seamlessly with the proposed role of the superior colliculus in target selection outlined in section 3.2. As noted there, the selection of a traget for action is the final event in the brain's real-time decision-making regarding "what to do next." The significance of gaze control, moreover, goes far beyond the matter of moving eyes-and-head in space. The gaze plays an organizing role in a wide range of behaviors by "leading" many forms of action, as has been shown in exquisite detail for manual reaching and manipulation (Johansson et al. 2001; see also Courjon et al. 2004; Jackson et al. 2005; Schneider &

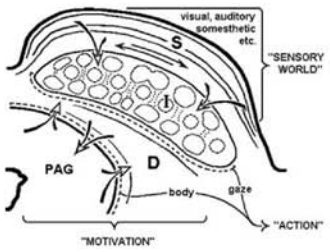

Figure 4. The three principal domains of "world" (target selection), "body" (action selection), and "motivation" (needs) that must interact to optimize decision processes in real time, portrayed in their proposed "primary" implementation in the roof of the midbrain. The extension of its logic into the forebrain, and the cerebral cortex of mammals in particular, can be conceived in terms of this primary system "writ large," as follows (cf. Fig. 6 in particular): A dorsolateral to ventromedial path from the surface of the colliculus to the midbrain aqueduct corresponds to a posterior to frontal to medial path in the cortex. In the reverse direction, and in functional terms, it reads "motivation," "action," and "world." **S**, **I**, and **D**: superficial, intermediate, and deep layers of the superior colliculus, respectively. **PAG**: the periaqueductal gray matter surrounding the midbrain cerebral aquaduct. Bidirectional arrow aligned with the collicular lamina stand for compensatory coordinate transformations. Drawing based in part on Harting et al. (1997).

Deubel 2002; Stuphorn et al. 2000; Werner et al. 1997). Nor is the output of the tecto-periaqueductal system limited to the species-specific orienting reflex: it includes escape behavior (Dean et al. 1989; Merker 1980; Sprague et al. 1961) as well as a number of innate postural schematisms associated with behaviors under periaqueductal control (Holstege et al. 1996; Lonstein et al. 1998).

In its primitive beginnings, the "world" of the proposed neural reality simulator presumably amounted to no more than a two-dimensional screen-like map of spatial directions on which potential targets might appear as mere loci of motion in an otherwise featureless noise field, defined more by their displacement than by any object features (see Stoerig & Barth 2001, for a plausible simulation). Advances on this primitive arrangement apparently proceeded by adding to it more sophisticated information from a rostral direction. Thus, the ability of a frog to side-step stationary barriers during prey-catching is dependent upon input to the tectum from the region of the caudal thalamus and pretectum, just anterior to the tectum (Ewert 1968; Ingle 1973). With the elaboration of the telencephalon, culminating in the neocortex of mammals, the arrangement was expanded further (see Section 4.5), into a fully articulated, panoramic three-dimensional world composed of shaped solid objects: the world of our familiar phenomenal experience.

4.3. Inhabiting a neural simulation

Whether primitive or advanced, the fundamental simplifying device of the proposed simulation space is to associate the origin of its shared body-world coordinate system for orienting with the head representation of its analog body. This does *not* mean that the coordinate system itself is head centered (i.e., moves with the head). At brainstem levels it appears, rather, to be oculocentric (Klier et al. 2001; Moschovakis 1996; Moschovakis & Highstein 1994). It means only that the coordinate system origin is lodged in the head representation of the simulated analog *visual* body, say in close proximity to its analog eye region. With such a location, a number of sensory-sensory mismatches and the contamination of sensory information by movement caused by the largely rotary displacements of eyes and head involved in perpetual orienting movements can be remedied – to a first approximation – by spherical coordinate transformations. This economy of control helps explain the fact that at the brainstem level not only eye movements, but also head movements, despite their very different musculo-skeletal demands, utilize a common intermediate control system organized in separate horizontal and vertical, that is, spherical, coordinates (Grobstein 1989; Masino 1992; Masino & Grobstein 1989; Masino & Knudsen 1990; see also Isa & Sasaki 2002). In humans, covert orienting of attention, as well as the visuomotor map for reaching (Gawryszewski et al. 2005; Vetter et al. 1999), appear to be framed in spherical coordinates,[3] perhaps reflecting collicular involvement in both functions (Müller et al. 2004; Werner et al. 1997).

There is reason to believe that the implicit "ego-center" origin of this coordinate space is the position we ourselves occupy when we are conscious, and that the analog body and analog world of that space is what we experience as and call our tangible, concrete body and the external world (cf. Note 2). This would explain the irreducible asymmetry adhering to the relation between perceiving subject and apprehended objects defining the conscious state. The ego-center places the conscious subject in an inherently "perspectival," viewpoint-based, relation to the contents of sensory consciousness. It is from there that objects are apprehended; objects do not apprehend the subject (cf. Merker 1997). By the same token, the one necessary constituent of consciousness that can never be an object of consciousness is that very vantage point itself, namely, the origin of the coordinate system of the simulation space. It cannot be an object of consciousness any more than an eye can see itself (Schopenhauer 1819, vol. 2, p. 491; see Baars 1988, pp. 327ff for this and other "contextual" aspects of consciousness).

Should these reasons appear somewhat abstract and rarefied, there is a far more concrete indication to the same effect. Our very body bears a tell-tale sign allowing us to recognize it as the product of a neural simulation. Vision differs topologically from somesthesis and audition by its limited angular subtense, particularly in animals with frontally directed eyes. The other two senses can be mapped *in toto* onto a spherical coordinate system for orienting, whereas vision is only partially mapped in this way. This is not in itself a problem, but becomes one given that vision can be directed, not only to the external world, but to the body itself. This necessitates some kind of junction or transition between the distal visual world and the proximal visual body, and there a problem does arise.

Though, as we have seen, the ego-center is present in consciousness by implication only, its location can be determined empirically (Cox 1999; Hering 1879/1942; Howard & Templeton 1966; Neelon et al. 2004; Roelofs 1959). It is single, and located behind the bridge of the nose inside our head. From there we *appear* to confront the visible world directly through an empty and single cyclopean aperture in the front of our head (Hering 1879/1942; Julesz 1971). Yet that is obviously a mere appearance, since if we were literally and actually located inside our heads we ought to see, not the world, but the anatomical tissues inside the front of our skulls when looking. The cyclopean aperture is a convenient neural fiction through which the distal visual world is "inserted" through a missing part of the proximal visual body, which is "without head" as it were or, more precisely, missing its upper face region (see Harding 1961). Somesthesis by contrast maintains unbroken continuity across this region. The empty opening through which we gaze out at the world betrays the simulated nature of the body and world that are given to us in consciousness. The essentials of the arrangement are depicted in highly schematic form in Figure 5.

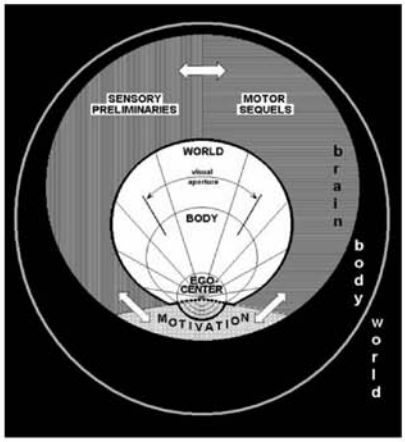

Figure 5. Highly schematic depiction of the nested relation between ego-center, neural body, and neural world constituting the analog neural simulation ("reality space") proposed as a solution to the tri-partite selection problem described in the text. Black depicts the physical universe, one part of which is the physical body (black oval), both of which are necessarily outside of consciousness. One part of the physical body is the physical brain (circle; shaded and unshaded). It contains the "reality space" of consciousness (unshaded), separated from other, nonconscious (shaded) functional domains by a heavy black line, signifying their exclusion from consciousness. Arrows mark interfaces across which neural information may pass without entering consciousness. The designation ego-center is a sensorimotor construct unrelated to the concept of self-consciousness. See text for further details.

The simulated nature of our body and world is further supported by a number of phenomena that alert us to the synthetic nature of what we typically take to be physical reality itself, that is, phenomena such as inattention blindness, change blindness, and allied effects (O'Reagan et al. 2000; Rensink 2002; Rensink et al. 1997; Simons & Chabris 1999). Such "deletions from consciousness" can be countered by appropriately placed microstimulation of the superior colliculus (Cavanaugh & Wurtz 2004; see also Müller et al. 2005). These various indications all support the conclusion that what we confront in sensory consciousness is indeed a simulated (synthetic) world and body.

As central residents of that simulation, we are subject to ever shifting moods, feelings, urges, emotions, and impulses. These, then, would be those aspects of the brain's motivational dynamics that reach consciousness (cf. Cabanac 1992; Panksepp 1982; 1998a). The reason they do so, according to the present proposal, is their relevance to the tripartite determination of what to do next, as outlined in the foregoing discussion. A striking illustration of this principle is afforded by respiratory control (Merker 2005). It is automatic and unconscious as long as partial pressures of blood gases stay within normal bounds, yet intrudes most forcefully on consciousness in the form of an acute sense of panic when they go out of bounds. Extreme blood gas values are an indication that urgent action on the environment – such as removing an airway obstruction or getting out of a carbon dioxide filled pit – may be imperative. That is what suddenly makes action selection and target selection relevant to respiratory control, which accordingly "enters consciousness" in the form of a powerful feeling of suffocation.

This example further illustrates the lack of any necessary connection between cognitive sophistication and the reason for something to enter consciousness. Even quite elementary functions may benefit from the efficiency provided by the triangular action-target-motivation interface of consciousness. It serves optimal decision-making in real time, on the broad front of its tripartite information base, concisely packaged in its multivariate simulation space. Such a utility is particularly valuable when a moment's hesitation may make a big difference in outcome, as in the suffocation example (but also in, say, agonistic encounters), quite apart from anything to do with advanced cognition. The evolution of such a utility could accordingly proceed independently of cognitive capacity, to crown the optic brain with its tectal machinery at the very outset of the vertebrate lineage.

In its peculiar nesting of a body inside a world, around an ego-center in a shared coordinate space subject to motivational bias, this interface possesses the essential attributes of phenomenal consciousness. As implemented in the midbrain and diencephalon, the arrangement is proposed to have served as the innate scaffolding supporting all further elaboration of conscious contents in phylogeny. Centered on the colliculus extending into periaqueductal gray, it will be further defined in section 4.5. A felicitous term for the functional state supported by the basic (mesodiencephalic) arrangement would accordingly be "primary consciousness" (Hodgson 1878; Petty 1998; Trevarthen & Reddy 2006).

4.4. Coherent, motivated behavior under sensory guidance in the absence of the cerebral cortex

The superordinate functional position attributed to mesodiencephalic mechanisms in previous sections of this article is supported by a number of empirical findings that receive a unified interpretation in this light. When the behavioral effects of local brain stimulation are systematically surveyed by means of depth electrodes, it is common to find that the most coherent, integrated, and natural-looking (whole, or "molar") behavioral reactions – be they orienting, exploration, or a variety of appetitive, consummatory, and defensive behaviors – are evoked by stimulation of diencephalic and midbrain sites, whereas stimulation at more rostral or caudal levels tends to evoke more fragmentary or incomplete behaviors (Adams 1979; Bandler & Keay 1996; Bard 1928; Brandao et al. 1999; Carrive et al. 1989; Fernandez de Molina & Hunsperger 1962; Hess 1954; Hess & Brugger 1943; Holstege & Georgiadis 2004; Hunsperger 1956; 1963; Hunsperger & Bucher 1967; Kaada 1951; Orlovsky & Shik 1976; Schaefer & Schneider 1968; Schuller & Radtke-Schuller 1990).

All of the behaviors just mentioned have also been exhibited by experimental animals after their cerebral cortex has been removed surgically, either in adulthood or neonatally. Best studied in this regard are rodents (Whishaw 1990; Woods 1964). After recovery, decorticate rats show no gross abnormalities in behavior that would allow a casual observer to identify them as impaired in an ordinary captive housing situation, although an experienced observer would be able to do so on the basis of cues in posture, movement, and appearance (Whishaw 1990; what follows relies on Whishaw's study, supplemented by additional sources as indicated). They stand, rear, climb, hang from bars, and sleep with normal postures (Vanderwolf et al. 1978). They groom, play (Panksepp et al. 1994; Pellis et al. 1992), swim, eat, and defend themselves (Vanderwolf et al. 1978) in ways that differ in some details from those of intact animals, but not in outline. Either sex is capable of mating successfully when paired with normal cage mates (Carter et al. 1982; Whishaw & Kolb 1985), though some behavioral components of normal mating are missing and some are abnormally executed. Neonatally decorticated rats as adults show the essentials of maternal behavior, which, though deficient in some respects, allows them to raise pups to maturity. Some, but not all, aspects of skilled movements survive decortication (Whishaw & Kolb 1988), and decorticate rats perform as readily as controls on a number of learning tests (Oakley 1983). Much of what is observed in rats (including mating and maternal behavior) is also true of cats with cortical removal in infancy: they move purposefully, orient themselves to their surroundings by vision and touch (as do the rodents), and are capable of solving a visual discrimination task in a T-maze (Bjursten et al. 1976; see also Bard & Bioch 1937).

The fact that coherent and well-organized molar behaviors are elicited by local stimulation in the mesodiencephalic region of intact animals and that coherent motivated behavior under environmental guidance is displayed spontaneously by animals lacking a cerebral cortex means that the neural mechanisms required to motivate, orchestrate, and provide spatial guidance for these behaviors are present in the parts of the brain that remain after decortication. Some aspects of these behaviors are dependent upon basal ganglia and basal forebrain functions remaining after the loss of their principal (cortical) source of afference (Wishaw 1990, p. 246), whereas the basic competences of decorticate animals reflect the capacity of upper brainstem mechanisms to sustain the global patterning, emotional valence, and spatial guidance of the postures and movements of orienting, defense, aggression, play, and other appetitive and consummatory behaviors (Adams 1979; Holstege & Georgiadis 2004; Masino 1992; Maskos et al. 2005; Panksepp 1982; Sakuma & Pfaff 1979; Swanson 2000). The particulars of the dependence of these behaviors on key structures located in the mesodiencephalic region has been repeatedly reviewed (Bassett & Taube 2001; Behbehani 1995; Groenewegen 2003; Haber & Fudge 1997; Horvitz 2000; Houk 1991; Jurgens 1994; Mouton 1999; Padel 1993; Panksepp 1998a; Prescott et al. 1999; Swanson 1987; 2000; ten Donkelaar 1988; Watt 2000; Watt & Pincus 2004; Winn 1998; Zahm 2006).

It is into the premotor circuitry of these ancient and highly conserved upper brainstem mechanisms that a wide range of systems place their bids for "where to look" and "what to do," irrespective of the level of sophistication of any one of these "bidding" systems. Each of them has independent access to effectors, and their upper brainstem interactions are not infrequently mediated by collaterals of such projections. The cerebral cortex is one prominent input to this system through the direct and indirect fiber projections emphasized in the foregoing discussion and sketched in Figure 3 (see also Swanson 2000). This relationship is, however, not a one-way affair. In fact, the manner in which the telencephalon is interfaced and integrated with the mesodiencephalic control system adds further definition to the central role of upper brainstem mechanisms in conscious functions.

4.5. Including the forebrain

Three cortical regions figure repeatedly and prominently in studies of cerebral mechanisms related to attention, neglect, and consciousness: namely, the posterior parietal cortex, the prefrontal cortex, and a medial territory centered on the cingulate gyrus (Baars et al. 2003, Fig. 1; Blumenfeld & Taylor 2003; Clower et al. 2001; Corbetta 1998; Han et al. 2003; Lynch et al. 1994; Mesulam 1999; Posner & Petersen 1990; Raz & Buhle 2006; Rees & Lavie 2001). A special connective and functional relationship exists between these three cortical territories and the mesodiencephalic system outlined in the foregoing discussion. It is most easily approached by considering their mutual interface in the nuclei of the dorsal thalamus. The latter can be divided into first-order (largely sensory relay) and higher-order ("association") thalamic nuclei (Sherman & Guillery 2001), and it is with the latter, *higher-order* nuclei, that the mesodiencephalic system maintains an intimate and complex relationship.

The two major higher-order nuclei of mammals are the mediodorsal nucleus, whose cortical projections define the prefrontal cortex, and the pulvinar complex related to a set of posterior cortical areas, including extrastriate visual

areas such as those of the posterior parietal cortex. Though proposed to serve as thalamic relays for cortico-cortical interactions (Sherman & Guillery 2001), these nuclei are not devoid of extra-telencephalic input, and both receive prominent input from the superior colliculus (Benevento & Fallon 1975; Harting et al. 1980; Lyon et al. 2005). Afferents to the pulvinar originate largely from the superficial collicular layers, whereas those destined for the mediodorsal nucleus are predominantly of intermediate layer origin. The latter projection targets a zone at the lateral edge of the mediodorsal nucleus related to the frontal eye fields (see Sommer & Wurtz 2004), the cortical territory most directly implicated in unilateral neglect of frontal origin (see Mesulam 1999, and references therein).

The cingulate gyrus, finally, is related to the mesodiencephalic system by its projections to the intermediate and deep layers of the colliculus (Harting et al. 1992; Sherman et al. 1979), the periaqueductal gray matter (An et al. 1998; Floyd et al. 2000), and by a conspicuously heavy projection to the *zona incerta* (Mitrofanis & Mikuletic 1999, Figs. 6 and 7). This latter structure is a mammalian derivative of the ventral thalamus of comparative terminology mentioned in section 4.1, and has emerged from obscurity only recently (see review by Mitrofanis 2005). It sends a topographically organized inhibitory projection to the superior colliculus, and reaches up into the thalamus above it to selectively innervate its higher-order nuclei bilaterally, likewise with powerful GABAergic inhibition (Barthó et al. 2002; Lavallée et al. 2005; Power et al. 1999; Trageser & Keller 2004).

Collicular input to the higher-order nuclei is excitatory, whereas their incertal input is inhibitory. This implies dynamic competition between colliculus and zona incerta for influence over the two principal thalamic dependencies of the prefrontal and the posterior parietal cortex. In this competition the inhibitory incertal element stands under cingulate cortex influence and is also in a position to inhibit the colliculus directly and with topographic specificity (Ficalora & Mize 1989; Kim et al. 1992; Ma 1996; May et al. 1997). These circumstances cannot but profoundly affect the functional dynamics of the three cortical territories with which we are concerned. The principal pathways relating them to the mesodiencephalic control system and the higher-order thalamic nuclei are depicted schematically in Figure 6.

Supplying a key node in the relations depicted in Figure 6, the zona incerta is monosynaptically (and often reciprocally and bilaterally) connected with on the order of 50 separate structures along the entire length of the neuraxis from spinal cord to olfactory bulb (my own conservative inventory of the literature, not counting connections with individual cortical areas separately). Internally, the zona incerta features profuse mutual connectivity in a setting of cytoarchitectonic and cytological heterogeneity in which GABAergic cells are prominent (Benson et al. 1991; 1992; Nicolelis et al. 1992; see Power & Mitrofanis 1999; 2001; and Bartho et al. 2002, p. 1002, for connective details). A combination of reciprocal external connectivity with internal mutual inhibition is the theoretically optimal solution for implementing global competitive interaction among structures separated by long distances (for background, see McFarland 1965; Snaith & Holland 1990; Prescott et al. 1999, pp. 27–29). The zona incerta accordingly may implement such a

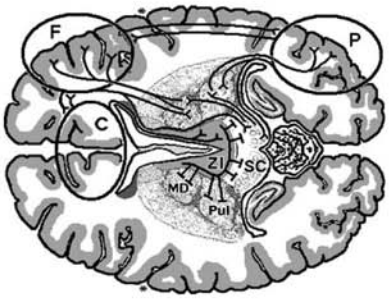

Figure 6. Composite diagram illustrating the interface between the mesodiencephalic system and the thalamocortical complex. Principal pathways by which the superior colliculus and the zona incerta relate to one another, as well as to the dorsal thalamus and the cerebral cortex, are indicated in black heavy lines. Excitatory connections end in a "Y", inhibitory connections in a "T". Abbreviations: **P**: parietal; **F**: frontal; **C**: cingulate cortex; **SC**: superior colliculus; **ZI**: zona incerta; **Pul**: pulvinar complex; **MD**: mediodorsal nucleus of the thalamus. The central sulcus is marked by an asterisk. See text for further detail.

scheme, and is hereby proposed to do so, as schematically illustrated in Figure 7.

The zona incerta – or the ventral thalamus of non-mammals – thus supplies the integrative machinery of the optic brain with a connective hub that seems designed to conduct mutually inhibitory trials of strength among a truly diverse set of afferents. They include, but are not limited to, visual, auditory, somatosensory, vestibular (Horowitz et al. 2005), cerebellar, striatal, collicular, motor, and limbic ones. The outcome of the competition – a neural decision – is conveyed to the intermediate and deep layers of the superior colliculus by a topographically organized inhibitory projection, as already mentioned. The collicular return projection to the zona incerta – like that of many incertal afferents – is non-topographic, implying greater specificity of incertal influence over the colliculus than the reverse. At the same time, incertal inhibitory output ascends into the association nuclei of the dorsal thalamus, establishing the zona incerta as a connective bridge straddling the mesodiencephalic and the thalamocortical systems.

Coupled with the scope of its connectivity along the neuraxis, this nodal position of the zona incerta lends it a potentially strategic role as an arbiter of moment-to-moment decision-making "in the light of all available evidence." As in the case of collicular target selection, the loss of such a high-level function need not generate conspicuous behavioral deficits, and does not appear to do so in rats with incertal lesions (Thompson & Bachman 1979). Rather, it would be expected to issue in suboptimal levels of resource allocation relative to shifting patterns of multiply interacting opportunities and needs. Preliminary indications regarding the great diversity and complexity of neuronal response properties in the zona incerta are worthy of note in this connection (Crutcher

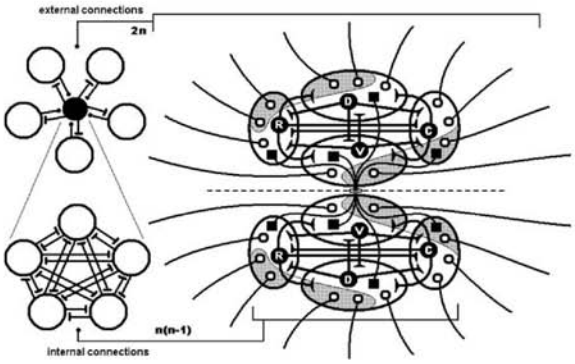

Figure 7. Schematic diagram illustrating zona incerta connectivity to the rest of the brain and of its subdivisions to one another, depicted bilaterally to emphasize the prominent commissural, "midline-straddling," nature of incertal connectivity. Ovals represent the principal incertal subnuclei, shown physically separated for graphical clarity. Labels **R**, **D**, **V**, and **C** mark the rostral, dorsal, ventral, and caudal subnuclei, respectively, and connections among them. Filled squares indicate that each subnucleus projects to all subnuclei on the opposite side. Open circles stand for incertal connections with the rest of the brain (typically reciprocal in nature). Shaded regions stand symbolically for the fact that functionally defined subregions of the zona incerta (sensory modalities, motor, limbic, etc.) often cut across its nuclear subdivisions. Inserts on the left summarize the two connective schemes that appear to be combined in the zona incerta. Long-distance (external) connections, for which wiring-efficiency is at a premium, connect n entities to a central hub (the zona incerta itself) by 2n (i.e., reciprocal) connections (expandable to new targets at the low cost of 2). Internal connectivity within the zona incerta (wiring efficiency *not* at a premium), by contrast, appears to follow the n(n – 1) connective principle (lower diagram, expanding the filled central circle of the upper diagram). The scheme as a whole idealizes evidence supplied by the pioneering studies of Mitrofanis and colleagues (references in the text), and awaits refinement in the light of further detailed studies.

et al. 1980; Kendrick et al. 1991; Ma 1996; Mungarndee et al. 2002; Nicolelis et al. 1992).

Finally, the zona incerta lies in immediate anterior continuity with the prerubral field and rostral interstitial nucleus of the medial longitudinal fasciculus, that is, with the rostral-most pole of the intermediate control system for orienting organized in spherical coordinates, mentioned in section 4.3. This rostral pole is specialized for vertical movement, whereas the system's horizontal components are found farther caudally, in paramedian reticular structures extending into the pons. Could it be that the zona incerta supplies a kind of origin for this coordinate system, a midline-straddling point of unity connected directly and via the colliculus to the rest of the coordinate space (Giolli et al. 2001; Kolmac et al. 1998; Leichnetz et al. 1987)? Incertal omnipause neurons are at least compatible with such an eventuality (Hikosaka & Wurtz 1983; Ma 1996). Nothing would be more elegant than to entrust the final arbitration of "what to do next" to a self-inhibitory "winner-take-all" or other decision network (Richards et al. 2006) lodged at the origin of the coordinate system that controls the orienting movements which execute that decision once made. As a primary perspectival viewpoint charged with changing motives, it would possess the essential attributes of a self (see sect. 4.3). Prominent incertal afference from cingulate cortex would fit such a role (cf. Northoff et al. 2006 for medial cortex and self), but short of further evidence, the suggestion must remain speculative.

4.5.1. Collicular gamma oscillations and cortical "binding.". The superior colliculus is the only place outside of the cerebral cortex in which fast oscillations in the gamma range have been shown to occur and to behave in a manner paralleling in all significant respects that of the cortex (Brecht et al. 1998; 1999; 2001). At the cortical level such oscillatory activity has been proposed to serve a "binding" function for consciousness (in the sense of integrating disparate elements of unitary conscious percepts) on circumstantial grounds (Engel et al. 1999; Engel & Singer 2001; Singer 2001). As we shall see, one need not, however, ascribe a unique role to gamma oscillations in either binding or consciousness to recognize that they may have consequences for cortico-collicular integration nevertheless.

Though sometimes portrayed as "the" problem of consciousness, the acuteness of the cortical binding problem must not be exaggerated. The pyramid architecture of point-to-point interareal connectivity within topographically organized cortical sensory domains ensures that corresponding points on areal topographies featuring different functional content (e.g., contour and color) are *connectively* and thus coherently related, even though the areas themselves occupy separate locations in the cortical sheet (Felleman & VanEssen 1991; cf. Fig. 2 and Note 2 of Merker 2004a).

The laminar superposition of numerous cortical areas in the colliculus takes this principle further. Here the joining of corresponding points on different cortical maps takes

place by direct laminar superposition of topographic projections of different cortical areas within a unified collicular topography. Thus, the output of different cortical areas are brought within the compass of the dendritic trees of single collicular neurons, which often straddle collicular laminar boundaries (Albers & Meek 1991; Laemle 1983; Langer & Lund 1974; Ma et al. 1990). Tight temporal synchrony of neuronal firing in separate cortical loci (through coupling to gamma oscillations) increases the probability that their joint activity will fall within the temporal window of integration of any neuron – whether cortical or subcortical – to which they project convergently (Abeles 1982; König et al. 1996). Synchronous activation of corresponding loci on separate cortical maps would accordingly assist such activity in crossing collicular thresholds by summation via the dendritic trees of convergently innervated collicular cells.

In crossing the collicular threshold – whether assisted by gamma synchrony or not – cortical activity would gain access to the mesodiencephalic system in all of its ramifications, projections to the cortex included (see Fig. 6). This, according to the present account, would be a principal step by which such activity enters awareness. If so, it follows that *one conscious content will not be replaced by another without involvement of the mesodiencephalic system (centered on the superior colliculus) as outlined here, even when that change is unaccompanied by eye movements.* This prediction is specific to the present perspective, and accordingly renders it testable. The means for doing so are exemplified by a recent functional imaging study of a visual-auditory illusion in humans (Watkins et al. 2006). That study revealed collicular activation associated with awareness of the illusion, though stimuli were identical on trials in which the illusion was not perceived, and central fixation was maintained throughout, confirming the prediction just made, in this particular instance.

This, then, would be the identity of the so far unidentified threshold featured in a recent programmatic proposal regarding conscious function (Crick & Koch 2003). Its identification with the threshold for access to the mesodiencephalic system centered on the colliculus (Figs. 4 & 6) is reinforced by the fact that layer V pyramidal cells supply the sole cortical projection to the colliculus. These cells exhibit a number of notable specializations: they do *not* give off collaterals to the thalamic reticular nucleus on passing through it (Jones 2002), their local intra-cortical connectivity appears stereotyped (Kozloski et al. 2001), and their apical dendrites branch in cortical layer I and carry specialized conductance mechanisms activated by top-down (feedback) connections in the superficial cortical layers (Larkum et al. 2004). This may ensure that activation of both the feedforward and feedback cortical system is typically required for the cortico-mesencephalic threshold to be crossed, such concurrent activation having been proposed as an essential condition for cortical information to reach awareness (Lamme & Spekreijse 2000; see also Merker 2004a, p. 566).

4.5.2. Consciousness and cortical memory. Penfield and Jasper proposed a role for the centrencephalic system in both consciousness and the laying down of cortical memories across the life span. A rationale for such a memory role is suggested by the present perspective. The perpetual and cumulative nature of cortical memory recording (Merker 2004a; 2004b; Standing 1973) puts a premium on economy of storage, that is, on concentrating memory recording to significant information (Haft 1998). A criterion for doing so is available in the system of integration for action as outlined here: Information that is important enough to capture control of behavior (i.e., by triggering an orienting movement placing its target in focal awareness) is also important enough to be consigned to permanent cortical storage. The focal presence of the target obviously will be the greater part of ensuring such an outcome, but it is likely to be actively supported as well by the system of dual colliculo-thalamic relays to cortex (cf. Fig. 6). From its parietal and frontal target areas, accessed in part via so-called matrix cell projections from the thalamus to the superficial cortical layers (Jones 1998), the mesodiencephalic influence would then propagate and spread through the cortex via intracortical top-down feedback connectivity.

The evidence for a "general learning system" (which includes the superior colliculus: Thompson 1993), mentioned in the introduction to section 3, would seem to bear on this proposal, as well. In fact, the severe capacity limitations of so called working memory (Baddeley 1992; Cowan 2001; Mandler 1975) are likely to derive in large part from the mesodiencephalic bottleneck which all attended (i.e., conscious) information must access according to the present proposal, just at the point where the parallel distributed data format of the forebrain *requires* conversion to a serial, limited capacity format to serve behavior.

4.5.3. The zona incerta and the seizures of absence epilepsy. It is to be noted, finally, that the Penfield and Jasper postulation of a centrencephalic system *symmetrically related to both cerebral hemispheres* was motivated in part by observations on the generalized seizures of absence epilepsy. The zona incerta sends a rich complement of commissural fibers across the midline not only to itself, but also to the association nuclei of the dorsal thalamus (Power & Mitrofanis 1999; 2001). It is also a prime locus for the induction of generalized epileptic seizures, being more sensitive than any other brain site to their induction by local infusion of carbachol (Brudzynski et al. 1995; see also Gioanni et al. 1991; Hamani et al. 1994). A number of phenomena that may accompany absence seizures can be readily related to the zona incerta. Thus, a forward bending or dropping of the head (or bending of the whole body to the ground; Penfield & Jasper 1954, p. 28) may relate to the already mentioned fact that the transition between the zona incerta and midbrain contains mechanisms for vertical control of eyes and head (Holstege & Cowie 1989; Waitzman et al. 2000; cf. sect. 4.2). The fluttering of the eyelids that often occurs in the same situation is also easily accommodated by the functional anatomy of this region (Morcuende et al. 2002; Schmidtke & Buttner-Ennever 1992).

The Penfield and Jasper definition of their proposed centrencephalic system always included explicit reference to the midbrain reticular formation. The zona incerta resembles a forward extension of the midbrain reticular formation beneath the thalamus (Ramón-Moliner & Nauta 1966), and much of the functional anatomy of the diencephalon needs to be re-examined in light of its

unusual connectivity. As noted by Barthó et al. (2002), the identification of a second, incertal, source of GABAergic innervation of the dorsal thalamus, in addition to that of the thalamic reticular nucleus, necessitates a re-evaluation of the entire issue of the nature of thalamic involvement in seizure generation and oscillatory thalamocortical activity (McCormick & Contreras 2001; Steriade 2001). This is all the more so since the even more recent discovery of a third source of powerful GABAergic thalamic inhibition, originating in the anterior pretectal nucleus (Bokor et al. 2005). One need not, however, await the outcome of such re-examination to identify the zona incerta as the perfect anatomical center-piece for the Penfield and Jasper centrencephalic hypothesis, though its obscurity at the time kept it from being recognized as such.

5. Consciousness in children born without cortex

Anencephaly is the medical term for a condition in which the cerebral hemispheres either fail to develop for genetic-developmental reasons or are massively compromized by trauma of a physical, vascular, toxic, hypoxic-ischemic, or infectious nature at some stage of their development. Strictly speaking, the term is a misnomer. The brain consists of far more than cerebral hemispheres or prosencephalon, yet various conditions of radical hemispheric damage are historically labelled anencephaly. When the condition is acquired, for example, by an intrauterine vascular accident (stroke) of the fetal brain, the damaged forebrain tissue may undergo wholesale resorption. It is replaced by cerebrospinal fluid filling otherwise empty meninges lining a normally shaped skull, as illustrated in Figure 8. The condition is then called hydranencephaly (Friede 1989), and is unrelated to the far more benign condition called hydrocephalus, in which cortical tissue is compressed by enlarging ventricles but is present in anatomically distorted form (Sutton et al. 1980).

The loss of cortex must be massive to be designated hydranencephaly, but it is seldom complete (see Fig. 8). It typically corresponds to the vast but somewhat variable forebrain expanse supplied by the anterior cerebral circulation (Myers 1989; Wintour et al. 1996). Variable remnants of cortex supplied by the posterior circulation, notably inferomedial occipital, but also basal portions of temporal cortex, and midline cortical tissue along the falx extending into medial frontal cortex, may be spared. The physical presence of such cortical tissue, clearly visible in Figure 8, need not mean, however, that it is connected to the thalamus (white matter loss often interrupts the visual radiations, for instance) or that it is even locally functional. On autopsy, such tissue may be found to be gliotic on microscopic examination or to exhibit other structural anomalies indicating loss of function (Marin-Padilla 1997; Takada et al. 1989). As Figure 8 shows, most cortical areas are simply missing in hydranencephaly, and with them the organized system of cortico-cortical connections that underlie the integrative activity of cortex and its proposed role in functions such as consciousness (Baars et al. 2003; Sporns et al. 2000).

An infant born with hydranencephaly may initially present no conspicuous symptoms (Andre et al. 1975), and occasionally the condition is not diagnosed until several months postnatally, when developmental milestones are missed. In the course of the first year of life, which is often though not invariably difficult, these infants typically develop a variety of complications that always include motoric ones (tonus, spasticity, cerebral palsy), and often include seizures, problems with temperature regulation, reflux/aspiration with pulmonary sequelae, and other health problems occasioning medical emergencies and attended by a high mortality rate. Were

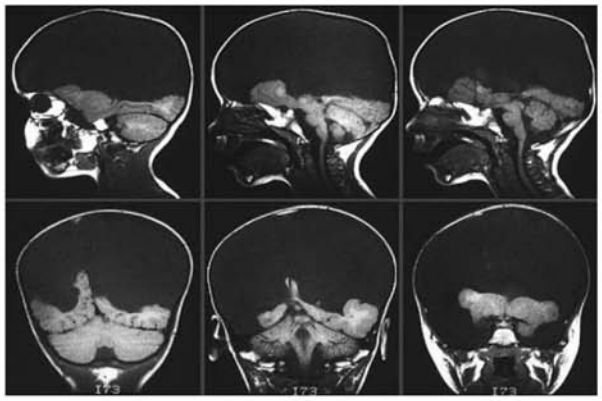

Figure 8. Saggittal and frontal magnetic resonance images of the head of a child with hydranencephaly. Spared ventromedial occipital and some midline cortical matter overlies an intact cerebellum and brainstem, whereas the rest of the cranium is filled with cerebrospinal fluid. Reprinted with the kind permission of the American College of Radiology (ACR Learning File, Neuroradiology, Edition 2, 2004).

one to confine one's assessment of the capacities of children with hydranencephaly to their presentation at this time – which for natural reasons is the period in the lives of these children to which the medical profession has the most exposure – it would be all too easy to paint a dismal picture of incapacity and unresponsiveness as the hydranencephaly norm. When, however, the health problems are brought under control by medication and other suitable interventions such as shunting to relieve intracranial pressure, the child tends to stabilize and with proper care and stimulation can survive for years and even decades (Counter 2005; Covington et al. 2003; Hoffman & Liss 1969; McAbee et al. 2000).

When examined after such stabilization has taken place, and in the setting of the home environment upon which these medically fragile children are crucially dependent, they give proof of being not only awake, but of the kind of responsiveness to their surroundings that qualifies as conscious by the criteria of ordinary neurological examination (Shewmon et al. 1999). The report by Shewmon and colleagues is the only published account based upon an assessment of the capacities of children with hydranencephaly under near optimal conditions, and the authors found that each of the four children they assessed was conscious. For detail, the reader is referred to the case reports included in the Shewmon et al. (1999) publication. Anecdotal reports by medical professionals to the same effect occasionally see print (Counter 2005), but compared to its theoretical and medical importance the issue remains woefully underexplored.

To supplement the limited information available in the medical literature on the behavior of children with hydranencephaly, I joined a worldwide internet self-help group formed by parents and primary caregivers of such children. Since February of 2003 I have read more than 26,000 e-mail messages passing between group members. Of these I have saved some 1,200 messages containing informative observations or revealing incidents involving the children. In October 2004 I joined five of these families for one week as part of a social get-together featuring extended visits to DisneyWorld with the children, who ranged in age from 10 months to 5 years. I followed and observed their behavior in the course of the many private and public events of that week, and documented it with four hours of video recordings.

My impression from this first-hand exposure to children with hydranencephaly confirms the account given by Shewmon and colleagues. These children are not only awake and often alert, but show responsiveness to their surroundings in the form of emotional or orienting reactions to environmental events (see Fig. 9 for an illustration), most readily to sounds, but also to salient visual stimuli (optic nerve status varies widely in hydranencephaly, discussed further on). They express pleasure by smiling and laughter, and aversion by "fussing," arching of the back and crying (in many gradations), their faces being animated by these emotional states. A familiar adult can employ this responsiveness to build up play sequences predictably progressing from smiling, through giggling, to laughter and great excitement on the part of the child. The children respond differentially to the voice and initiatives of familiars, and show preferences for certain situations and stimuli over others, such as a specific familiar toy, tune, or video program, and

Figure 9. The reaction of a three-year-old girl with hydranencephaly in a social situation in which her baby brother has been placed in her arms by her parents, who face her attentively and help support the baby while photographing.

apparently can even come to expect their regular presence in the course of recurrent daily routines.

Though behavior varies from child to child and over time in all these respects, some of these children may even take behavioral initiatives within the severe limitations of their motor disabilities, in the form of instrumental behaviors such as making noise by kicking trinkets hanging in a special frame constructed for the purpose ("little room"), or activating favorite toys by switches, presumably based upon associative learning of the connection between actions and their effects. Such behaviors are accompanied by situationally appropriate signs of pleasure or excitement on the part of the child, indicating that they involve the kind of coherent interaction between environmental stimuli, motivational-emotional mechanisms, and bodily actions for which the mesodiencephalic system outlined in this article is proposed to have evolved. The children are, moreover, subject to the seizures of absence epilepsy. Parents recognize these lapses of accessibility in their children, commenting on them in terms such as "she is off talking with the angels," and parents have no trouble recognizing when their child "is back." As discussed earlier, episodes of absence in this form of epilepsy represent a basic affliction of consciousness (cf. Blumenfeld & Taylor 2003). The fact that these children exhibit such episodes would seem to be a weighty piece of evidence regarding their conscious status.

In view of the functional considerations reviewed in the foregoing, none of these behavioral manifestations in children with hydranencephaly ought to occasion any surprise, and no special explanations such as neural reorganization based on plasticity are needed to account for them. Rather, they are what the nodal position of mesodiencephalic mechanisms in convergent neural integration, along with the comparative evidence regarding the behavior of mammals in the absence of cerebral cortex, would lead us to expect. Nor is there much warrant for attempting to attribute these behaviors to remnant cortical tissue. Besides the questionable functional status of spared cortex already alluded to, a significant functional asymmetry speaks directly against it. As common as it is for some occipital cortex to remain in these individuals, so is it rare for any auditory cortex to be spared. Yet, sensory responsiveness in hydranencephaly shows the opposite asymmetry: hearing is generally preserved, whereas vision tends to be compromised (Hydranencephaly Group Survey 2003). The pattern is easily accounted for by the intactness of the

brainstem auditory system in these children (Lott et al. 1986; Yuge & Kaga 1998), crowned by a projection from inferior to superior colliculus. By contrast, vision in these children is liable to be compromised already at the level of the optic nerve. The latter's blood supply through the anterior cerebral circulation exposes it to damage in hydranencephaly, and its status varies widely in affected children (Jones & France 1978).

What is surprising, instead, is the routine classification of children with hydranencephaly into the diagnostic category of "vegetative state" (Multi-Society Task Force 1994), apparently in conformity with a theoretical identification between the cortex as an anatomical entity and consciousness as a function. It is this very identification which has been under critical examination in the present target article. To the extent to which the arguments and the evidence presented here have any merit, such an identification is not tenable, and the routine attribution of a lack of awareness to children lacking cortex from birth would accordingly be inadmissible. The extent of awareness and other capacities in these children must be based on assessment in its own right, by appropriate neurological tests, and not by reference to the status of their cortical tissue (Shewmon 2004). Moreover, considering the medically fragile status of many of these children, such behavioral assessment must be performed under optimal circumstances.

Properly assessed, the behavior of children with early loss of their hemispheres opens a unique window on the functional capacities of a human brainstem deprived of its cerebral cortex early in intrauterine development. They tell us, for one thing, that the human brainstem is specifically human: these children smile and laugh in the specifically human manner, which is different from that of our closest relatives among the apes (Provine & Yong 1991; van Hooff 1972). This means that the human brainstem incorporates mechanisms implementing specifically human capacities, as shown long ago by the neurologist Gamper on the basis of his detailed cinematographically documented account of a congenitally anencephalic girl entrusted to his care (Gamper 1926). In her case, there is no possibility that remnant hemispheric tissue might account for her human smile, since detailed postmortem histology disclosed that she had no neural tissue above the level of the thalamus, and even her thalamus was not functional.

The implication of the present account is that unless there are further complications, such a child should be *expected* to be conscious, that is, possessed of the primary consciousness by which environmental sensory information is related to bodily action (such as orienting) and motivation/emotion through the brainstem system outlined in the foregoing. The basic features of that system evolved long before the cerebral hemispheres embarked on their spectacular expansion in mammals to supply it with a new form of information based upon cumulative integration of individual experience across the lifetime (see Merker 2004a). Now as then, this brainstem system performs for the cortex, as for the rest of the brain, a basic function: that of integrating the varied and widely distributed information needed to make the best choice of the very next act. That function, according to the present account, is the essential reason for our being conscious in the first place. The integrated and coherent relationship it establishes between environmental events, motivation/emotion, and actions around the pivotal node of an egocenter would seem to offer a definition of a "being" in biological terms.

6. Implications for medical ethics

Needless to say, the present account has ramifying implications for issues in medical ethics. One of these concerns pain management in children with hydranencephaly and similar conditions. It is not uncommon for parents to encounter surprise on the part of medical professionals when requesting analgesia or anesthesia for their crying child during invasive procedures, a situation in some ways reminiscent of what was found in the case of neonates only a few decades back (Anand & Hickey 1987). They also extend to more general issues pertaining to the quality of care appropriate to these children, and ultimately to questions such as the meaning of personhood and even medical definitions of death (see, e.g., Shewmon et al. 1989, and references therein). Such questions are decidedly beyond the scope of the present article, which is meant only to raise those issues of a theoretical and empirical nature which are prior to and essential for finding reasoned and responsible answers to the ethical ones. Suffice it to say that the evidence surveyed here gives no support for basing a search for such answers on the assumption that "awareness," in the primary sense of coherent relatedness of a motivated being to his or her surroundings, is an exclusively cortical function and cannot exist without it.

7. Conclusion

The evidence and functional arguments reviewed in this article are not easily reconciled with an exclusive identification of the cerebral cortex as the medium of conscious function. They even suggest that the primary function of consciousness – that of matching opportunities with needs in a central motion-stabilized body–world interface organized around an ego-center – vastly antedates the invention of neocortex by mammals, and may in fact have an implementation in the upper brainstem without it. The tacit consensus concerning the cerebral cortex as the "organ of consciousness" would thus have been reached prematurely, and may in fact be seriously in error. This has not always been so, as indicated by the review of the Penfield and Jasper (1954) "centrencephalic" theory of consciousness and volitional behavior with which we began. As we have seen, their proposal has not only been strengthened by certain findings accumulating since it was first formulated more than half a century ago, but, suitably updated, it still appears capable of providing a general framework for the integration of a vast array of diverse facts spanning from the basics of the vertebrate brain plan to evidence for awareness in children born without a cortex. Whether such a framework can be developed into a comprehensive account of the neural organization of consciousness will depend upon resolving a number of the empirical and theoretical questions left unanswered in the foregoing discussion. Preliminary though it may be, that discussion suggests that part of the endeavor to resolve these questions will require close scrutiny of conserved and convergently innervated upper

brainstem mechanisms as potential key components of a neural mechanism of consciousness.

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NOTES

1. In what follows, the term "cortex" will always be taken to mean all or part of the cerebral cortex along with its associated dorsal thalamic and claustral nuclear apparatus. The thalamic reticular nucleus, being functionally intrinsic to this thalamocortical complex is regarded as being part of it despite its embryological and phylogenetic origin in the ventral thalamus (it is directly continuous with the lateral margin of the zona incerta). Unless otherwise indicated, "subcortical" will refer to all central nervous system tissue that is not thalamocortical complex in this sense, and "brainstem" will refer to diencephalon and the rest of the entire neuraxis caudal to it.

2. To avoid possible misunderstanding of this key point, note that the analog "reality simulation" proposed here has nothing to do with a facility for simulating things such as alternate courses of action by, say, letting them unfold "in imagination," or any other version of an "inner world," "subjective thought," "fantasy," or the like. Such capacities are derivative ones, dependent upon additional neural structures whose operations presuppose those described here. The purpose of the "analog simulation" defined here is first and foremost to veridically reflect states of the world, the body, and needs at whatever level of sophistication a given species implements those realities. It is thus most directly related to the model of Philipona and colleagues (2003; 2004), as well as to the "situation room analogy" developed by Lehar (2002).

3. Note that in some of the animal and human studies cited in this passage the term "Cartesian" occurs as a misnomer for "spherical." They all refer to a system organized in terms of "azimuth" and "elevation," that is, a system of spherical coordinates.

Open Peer Commentary

The mesencephalon as a source of preattentive consciousness

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Abstract: By themselves, mesencephalic subcortical mechanisms provide a preattentive kind of consciousness, related to stimulus-related, short-latency dopamine release triggered by collicular input. Elaborate forms of consciousness, containing identifiable objects (visual, auditory, tactile, or chemical), imply longer-lasting phenomena that depend on the activation of prosencephalic networks. Nevertheless, the maintenance of these higher-level networks strongly depends on long-lasting mesencephalic dopamine release.

Following and expanding on Penfield's (1952) and Thompson's (1993) ideas, Merker's provocative article proposes a central role of the upper brainstem in the mechanisms of consciousness, while the telencephalon and diencephalon serve as a medium for the increasing elaboration of conscious contents. The sensorimotor, multimodal integrative role of the brainstem is supported by large amounts of evidence, and few would argue against its key role in behavioral organization. Merker goes beyond this conception by proposing a "selection triangle," based on action selection (substantia nigra, SN), target selection (superior colliculus, SC) and motivational rating (periaqueductal gray), that controls telencephalic processing, serves to regulate behavior, and implies a conscious mode of function. In a rudimentary form, this system might be present in the earliest chordates, while the evolutionary development of the telencephalon has served to provide plasticity and to expand this system by virtue of parallel processing. An intriguing element in Merker's proposal is the role of the *zona incerta*, a GABAergic complex that is suggested to operate in competition with the SC for control of higher cortical areas.

There is no doubt that further research is necessary regarding the role of subcortical structures in conscious experience and cognitive processing in general. Cognitive neurosciences have been excessively focused on the cerebral cortex as the neural foundation of all higher psychological functions. Merker's article clearly suggests that subcortex also plays an important role deserving investigation. The compelling evidence reviewed in the target article could be not only a good inducement, but also a starting point for such research.

Our commentary is focused on the role of the midbrain superior colliculus and mesencephalic dopaminergic nuclei in orienting and goal-directed behavior (Aboitiz et al. 2006). From being originally considered to be a system that codifies reward, subsequent studies emphasized the role of the dopaminergic (DA) system in several functions like alertness, reward prediction, attention, and working memory. Behavioral and physiological approaches suggest that there are two modes of DA signaling. Tonic, longer lasting DA release may be more related to the maintenance of a goal representation in working memory, and to sustained attention during the execution of behavior (Bandyopadhyay et al. 2005; Muller et al. 1998; Rossetti & Carboni 2005; Zhang et al. 2004). On the other hand, short-latency, phasic, stimulus-related DA release (SRDR; 70–100 ms post stimulus latency, <200 ms duration) is related to unpredicted, salient stimuli and participates in updating goal representations, in attentional shifts, and in reward prediction (Montague et al. 2004; Phillips et al. 2003; Redgrave & Gurney 2006). The balance between these two systems is crucial, as failure to maintain the behavioral goal results in distractibility, and failure to update it with new sensory evidence results in perseverance (Aboitiz et al. 2006).

Several lines of evidence point to the deep layers of the superior colliculus (SC) as the main source of short-latency sensory input into the substantia nigra, be it in the context of orienting behavior toward visual stimuli (Coizet et al. 2003; Comoli et al. 2003; Dommett et al. 2005; Redgrave & Gurney 2006) or avoidance behavior in response to noxious stimuli. In the second case, aversive stimuli elicit a short-latency (<100 ms) phasic DA suppression (Ungless et al. 2004). In some contexts, SRDR works as a reward prediction device, selecting behaviors that maximize future rewards (Montague

et al. 2004; Schultz & Dickinson 2000; Tobler et al. 2003, Waelti et al. 2001), which is in accordance with the "action-selection" role for the SC and SN proposed by Merker.

However, in real-life conditions, the reward value of many unexpected events is unknown at the time that SRDR takes place (Redgrave & Gurney 2006). These authors consider that, perhaps more than predicting the occurrence of reward, SRDR has a role in the recollection of actions that triggered an unpredicted event. In other words, every time a salient, unexpected stimulus is produced, SRDR in the corpus striatum, amygdala, and prefrontal cortex allows an association of the sensory, motor, and contextual situations immediately previous to this event, so that the animal may develop a "causative theory" of the events that led to this unpredicted stimulus and will become able to generate them in the future (Redgrave & Gurney 2006). If this stimulus is subsequently associated with positive or negative reinforcement, the animal will know what to do in order to approach or avoid this situation, respectively.

Besides the association with contextual information, what kind of knowledge about the unpredicted stimulus itself does the animal obtain from SRDR? If the primary short-latency input to the SN is the SC, it cannot be much. Visually, mammalian collicular neurons tend to respond to spatially localized changes in luminosity that signal movement or appearance or disappearance of objects in the visual field, while being relatively insensitive to object-specific characteristics (Sparks & Jay 1986, Wurtz & Albano 1980). Furthermore, SRDR is considered to relate to pre-saccadic processing in which attention is deviated to the unattended salient event, and there is not much information about the appetitive or aversive reinforcement consequences (reviewed in Redgrave & Gurney 2006).

In agreement with Merker's proposal, conscious experience may take place in preattentive (presaccadic) stages (Koch & Tsuchiya 2007). Nevertheless, we may ask the question about what contents might this conscious function have at the collicular level. Visually, object-relevant evidence may not be fully available at this point, and it is difficult to think of a conscious process without identifiable beings or objects in it. In our view, the role of mesencephalic, subcortical mechanisms in consciousness might be better described as providing a sort of "preattentive/presaccadic conscious state," related to alertness, attentional shifts, and decision making. The participation of higher telencephalic centers is necessary to make this a sustained event into which short-term memory may participate, thus providing the essential, recursive character of higher consciousness. In this context, the longer-lasting, sustained dopamine release that supports attention and working memory may contribute to the maintenance of this kind of perception online in higher telencephalic components in order to achieve goals that are not immediately available (Aboitiz et al. 2006). In other words, Merker is quite right in assigning the mesencephalic-basal forebrain level an important role in primitive orienting and goal-directed control, which serves as a basis for a primordial, preattentive form of consciousness; but the higher telencephalic centers are necessary for the elaboration of more complex forms of behavior and recursive, object-related consciousness.

Consciousness, cortical function, and pain perception in nonverbal humans

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Abstract: Postulating the subcortical organization of human consciousness provides a critical link for the construal of pain in patients with impaired cortical function or cortical immaturity during early development. Practical implications of the centrencephalic proposal include the redefinition of pain, improved pain assessment in nonverbal humans, and benefits of adequate analgesia/anesthesia for these patients, which certainly justify the rigorous scientific efforts required.

A reappraisal of the mechanisms of human consciousness, differentiating it from its attributes, functions, or contents, is long overdue. Widely held concepts about the key mechanisms of consciousness, or its fullest expression via the human brain, have not been reexamined in the light of accumulating evidence since the 1970s. Merker presents the organization of a subcortical system (the centrencephalic system proposed by Penfield and Jasper in the 1950s; see, e.g., Penfield & Jasper 1954), with multiple lines of anatomical, neurophysiological, behavioral, clinical, and neuropathological evidence, and a teleological rationale – all of which support a persuasive argument for the subcortical control and temporal sequencing of behavior. Advanced neuroimaging techniques or other tools can now be applied to testable hypotheses derived from the updated centrencephalic theory, an evaluation not possible 50 years ago. One distressing impact of associating consciousness with cortical function, briefly mentioned by Merker in section 6 of the target article, pertains to the mistaken notions regarding pain perception in patient populations with impaired cortical function or cortical immaturity. Because of its clinical, ethical, and social importance, this commentary focuses on the impact of centrencephalic theory on the capacity for pain perception in subjects with impaired cortical function or cortical immaturity during early development.

Despite a higher prevalence of pain in patients with impaired cortical function (Breau et al. 2004; Ferrell et al. 1995; Parmelee 1996; Stallard et al. 2001), such patients – not unlike the children with hydranencephaly described by Merker – receive fewer analgesics as compared with matched cognitively intact patients (Bell 1997; Feldt et al. 1998; Koh et al. 2004; Malviya et al. 2001; Stallard et al. 2001). Geriatric patients with dementia also receive fewer and lower doses of opioid or nonopioid analgesics than those received by comparable, but cognitively intact elders (Bell 1997; Closs et al. 2004; Feldt et al. 1998; Forster et al. 2000; Horgas & Tsai 1998). When we consider cortical immaturity during early development, the impact of these practices appears even greater. Human neonates, preterm and full-term, were previously thought to be insensitive to pain and were routinely subjected to surgical operations without adequate anesthesia or analgesia (Anand & Aynsley-Green 1985; Anand & Carr 1989). Large numbers of newborn infants are currently exposed to painful invasive procedures without appropriate analgesia (Johnston et al. 1997; Porter & Anand 1998; Simons et al. 2003) and recent reviews have questioned the ability of premature newborns or fetuses to experience pain (Derbyshire 2006; Lee et al. 2005; Mellor et al. 2005). Clinical practices denying or discounting the pain experienced by those who have little or no self-report recapitulate the opinions of leading physicians in 19th-century America, as, for example, when "Dr. Abel Pierson, Henry J. Bigelow, and others, ...assumed that the ability to experience pain was related to intelligence, memory, and rationality; like the lower animals, the very young lacked the mental capacity to suffer" (Pernick 1985).

The primary reasons for disregarding the experience of pain in those with limited cortical function include the current definition of pain and the exclusive association of human consciousness with cortical function.

Within the medical/scientific community, concepts of pain are based on its semantic definition rather than the actual experience it signifies. Pain is defined by Merskey and Bogduk (1994) as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage," followed by the note that, "Pain is always subjective. Each individual learns the application of the word through

experiences related to injury in early life" (Merskey & Bogduk 1994). Over the years, this definition has propagated undue credibility for the verbal expression of pain, defined within the context of adult consciousness, engendering medical practices that regard verbal self-report as the "gold standard" for pain (K. D. Craig 1997; Cunningham 1998; 1999). Major flaws in this definition include its excessive reliance on verbal self-report, the criterion that some form of learning is required in order to experience pain, and its focus on use of this word rather than the experience of pain (Anand & Craig 1996; Anand et al. 1999; K. D. Craig 1997; Shapiro 1999; Wall 1997).

Confusion regarding pain perception in early life continues to hinge on various interpretations of this flawed definition (Benatar & Benatar 2001; Derbyshire 2006; Lee et al. 2005), generating a circular argument that "to experience pain, infants must first learn what is pain; to learn what pain is, they must first experience it." The experience of pain primarily informs conscious beings of bodily harm; its perception is vital to survival and cannot depend on putative memories of prior painful experiences (Anand et al. 1999; Cunningham 1999). Consistent with this rationale, even the first exposure to bodily injury demonstrates the clinical signs of pain, regardless of whether tissue damage occurs during fetal or neonatal life (Grunau & Craig 1987; Williams 2005). The experience of pain must precede any responses that ensue (verbal, behavioral, or physiological), whereas the relationships between feeling pain and reporting pain are highly context-dependent (Anand & Craig 1996; A. D. Craig 2003).

The entity of consciousness, as discussed in greater detail elsewhere (Anand et al. 1999; Benatar & Benatar 2001), is mistakenly equated with development of the human mind (Benatar & Benatar 2001; Cunningham 1998; Derbyshire 2006) and burdened with *"the expectation that living organisms must exhibit certain attributes or capabilities analogous to the adult human in order to fulfill the criteria for consciousness"* (Anand et al. 1999). Some authors argue that fetuses or neonates are not conscious, that they are complex automatons (Derbyshire & Furedi 1996; Lloyd-Thomas & Fitzgerald 1996; Zelazo 2004), simply manifesting various reflexes triggered by tissue injury, but incapable of experiencing pain because they lack consciousness or cortical maturity (Benatar & Benatar 2001; Derbyshire 2006; Lee et al. 2005; Mellor et al. 2005).

Closer examination reveals three major flaws in this scientific rationale. First, pain perception is portrayed as a 'hard-wired' system, passively transmitting pain impulses until "perception" occurs in the cortex (Derbyshire 2006; Lee et al. 2005; Mellor et al. 2005). Beginning from the Gate Control Theory of pain (Melzack & Wall 1965), accumulating evidence over the past 40 years should lead us to discard this view of pain.

Second, it assumes that fetal or neonatal pain perception must activate the same neural structures as in the adult; immaturity of these areas then supports the argument that fetuses or premature neonates cannot experience pain. However, multiple lines of evidence show that the structures used for pain processing in early development are unique and different from adults and that some of these structures/mechanisms are not maintained beyond specific developmental periods (Fitzgerald 2005; Narsinghani & Anand 2000). The immature pain system thus plays a crucial signaling role during each stage of development and therefore uses different neural elements available at specific times during development to fulfill this role (Glover & Fisk 1996).

Third, the immaturity of thalamocortical connections is proposed as an argument against fetal pain perception (Derbyshire 2006; Lee et al. 2005; Mellor et al. 2005). This reasoning, however, ignores clinical data showing that ablation or stimulation of somatosensory cortex does not alter pain perception in adults, whereas thalamic ablation or stimulation does (Brooks et al. 2005; A. D. Craig 2003; Nandi et al. 2003). The fetal thalamus develops much earlier than the cortex (Erzurumlu & Jhaveri 1990; O'Leary et al. 1992; Ulfig et al. 2000), supporting clinical observations of fetal behavior in response to tissue injury (Fisk et al. 2001; Williams 2005). Functionally specific cortical activity in response to tactile or painful stimuli in premature neonates (Bartocci et al. 2006; Slater et al. 2006) provides further evidence for the thalamocortical signaling of pain.

Functional development of the centrencephalic system very likely mediates the onset of consciousness in fetal life, defining the "being" in biological terms (Hepper & Shahidullah 1994 and Merker's target article), and enabling its responses to invasions of bodily integrity (Wall 1996, 1997).

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Theoretical sequelae of a chronic neglect and unawareness of prefrontotectal pathways in the human brain

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Abstract: Attention research with prefrontal patients supports Merker's argument regarding the crucial role for the midbrain in higher cognition, through largely overlooked and misunderstood prefrontotectal connectivity. However, information theoretic analyses reveal that both exogenous (i.e., collicular) and endogenous (prefrontal) sources of information are responsible for large-scale context-sensitive brain dynamics, with prefrontal cortex being at the top of the hierarchy for cognitive control.

In his target article Merker reminds us of the critical role of midbrain structures for higher cognition in humans. This timely reminder should renew the interest for the study of cortical–subcortical interactions underlying human cognition. Our own research on the attentional disorders in neurological patients, although partly consistent with Merker's claims, calls for a revision of the theoretical implications of the centrencephalic hypothesis in light of the superordinate position of prefrontal cortex in the functional hierarchy of control in the human brain (Barceló & Knight 2000; in press; Barceló et al. 2000; Fuster 1997). In his otherwise very thorough review of brain anatomy and function, Merker does not consider the existence of direct prefrontotectal pathways in the human brain (Figs. 4 and 6 of the target article). In our view, this piece of anatomy carries crucial implications for computing and interpreting information processing within the central nervous system.

Direct prefrontotectal pathways have remained relatively unexplored since their discovery in primates by Goldman-Rakic and Nauta (1976). Failure to notice the relevance of prefrontotectal pathways abounds even in authoritative reviews of prefrontal anatomy (Petrides & Pandya 2002), and consequently, the putative functions of such connectivity have been overlooked or downplayed by recent models about the neural control of human cognition (Miller & Cohen 2001; Posner & Petersen 1990). This route was originally thought to aid the tracking of visual targets in spatial coordinates and was related to the cortical control of visually guided saccades and visuospatial distractibility (Gaymard et al. 2003; Pierrot-Deseilligny et al. 1991). Only recently has this route been related to the top-down control of voluntary and goal-directed behavior (Barceló & Knight 2000; in press; Friston 2005; Munoz & Everling 2004). The dorsolateral

prefrontal region involved, which corresponds to the middle third of the principal sulcus in the monkey, has been shown to subserve not only spatial, but also more general working memory functions closely tied in with awareness (Petrides & Pandya 2002). Hence, it seems justified to ponder the role of prefrontotectal pathways in target and action selection (sects 3.2 and 4 of the target article). In contrast to Merker's proposal of an "*anatomically subcortical but functionally supra-cortical*" system, we argue that prefrontotectal pathways evolved to allow the human prefrontal cortex to control the centrencephalic system, in line with the evolution of control architectures in the nervous system (cf. Fuster 1997).

Our argument can be substantiated by the extensive research on the neural bases of selective attention (i.e., orienting) to spatial, target, and task-set information. Most evidence for a collicular implication in target selection revolves around the selection of *the spatial location* of relatively novel, salient, or distinct perceptual objects whose abrupt onset triggers sensory and motor adjustments collectively known as an *orienting response* (Sokolov 1963). A cortical marker of the orienting response can

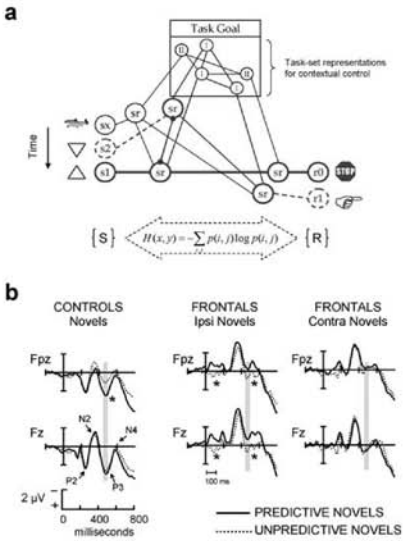

Figure 1 (Barceló & Knight). Hypothetical prefronto-tectal interactions during visual orienting to familiar and novel task-set information. (**a**) Information theoretic model of prefrontal function (adapted from Miller & Cohen, 2001). The neural representation of pools of stimulus features {S} and motor responses {R} are connected through several hierarchical levels of intervening sensorimotor processes in the central nervous system (cf. Fuster 1997). Familiar and well-rehearsed visual discriminations between upright (distracters) and upside-down (target) triangles rapidly and randomly flashed to both visual hemifields require sustained maintenance of a superordinate task-set representation (*task-set I*). This higher task-set representation holds other subordinate sensorimotor units (*sr*) in an active state at subcortical and/or posterior cortical structures, thus providing intervening pathways between perceptual and motor units. Lateral prefrontal cortex has been proposed to hold superordinate contextual representations in working memory (Miller & Cohen 2001). The onset of a familiar event triggers the updating of its corresponding sensory (*s1, s2*) and sensorimotor units (*s1-r0, s2-r1*) at subcortical and/or posterior cortical structures, without modifying the superordinate representation of familiar information. On the contrary, task-irrelevant unexpected novel events (*sx*) trigger an orienting response that demands updating of the active superordinate representation of task-set information (to new *task-set II*). The novel task-set II competes for attentional resources with the familiar task-set I, thus causing behavioral conflict and distractibility. When the novel event predicts the appearance of a target event in a predictable context, then a momentary conflict between two superordinate task-sets rapidly turns into anticipatory activation of the familiar task-set I, resulting in an amelioration of behavioral distractibility. (**b**) The cortical marker of the orienting response to unpredictive and predictive novel events displayed at the ipsi- and contralesion visual hemifields of patients with unilateral lesions to their dorsolateral prefrontal cortex (middle and right columns) are compared with data collapsed across both visual hemifields in controls (left column). Novel events evoked frontally distributed "novelty P3" potentials in Controls that were severely reduced in the Frontal patients regardless of the predictive value of the novel events or its visual hemifield of display. Importantly, predictive novels elicited anomalous sustained early 50–200 ms negativities over the lesioned prefrontal cortex (Ipsi Novels). The early timing of these negativities suggested conflict signals from prefrontotectal pathways that could not be dealt with because of missing superordinate task-set representations at prefrontal cortex. Grey bars indicate the time window for novelty P3 measurement. Fpz: Mid-frontopolar region; Fz: Mid-frontocentral region (for a full explanation of the task design, see Barceló & Knight 2000; Barceló et al. 2000).

be measured as a stereotyped scalp-recorded event-related potential, the so-called "novelty P3," which indicates that a novel event has captured attention and, at that point in time, is most likely within the focus of mind (Friedman et al. 2001). The novelty P3 potential depends on the integrity of a distributed cortical network including dorsolateral prefrontal, temporo-parietal, and mesial temporal cortices (Knight & Scabini 1998). This cortical marker of the orienting response was originally described as an involuntary reaction to novel and salient stimulation reflecting modality nonspecific cortical-subcortical interactions (i.e., visual novelty P3 activations do not follow the retinotopy of the geniculostriate pathways; cf. Sokolov 1963; Friston 2005), that most likely involve faster prefrontotectal pathways (see Fig. 1b; Barceló & Knight, in press). These cortical modulations could be likened to the property of the centrencephalic system of being "symmetrically related to both cerebral hemispheres" (sect. 3.2 of the target article). New task designs and an information theoretic analytical approach have revealed more top-down cortical control in this brain's orienting response than was originally suspected (see Figs. 1a, 1b; Barceló & Knight 2000, in press; Barceló et al. 2002, 2006).

Target and action selection require integration of contextual information across the spatio-temporal dimensions of our physical world. We orient to those targets that are perceptually salient or behaviorally relevant. However, the information content of a target for perception or action depends on the learned associations between exogenous sensory signals and past short- and long-term memories and plans of action. These context-dependent associations between sets of stimuli and responses for the accomplishment of internal goals are putatively encoded at hierarchically ordered levels of representation in the nervous system (Fig. 1a). Even if the centrencephalic system has direct control over sensory (i.e., *s1*, *s2*), motor (i.e., *r0*, *r1*), and some sensorimotor (*sr*) representations needed to perform simple and familiar visuospatial discriminations, it does not seem as well equipped as prefrontal cortex for accessing the short- and long-term memories necessary for the temporal organization of human behavior (Fuster 1997). The neural decisions about whether a novel sensory signal should be selected as a target (i.e., sensorimotor pathway *s2-r1* in Fig. 1a), or inhibited as a distracter (i.e., *s1-r0* in Fig. 1a), and whether these associations are to be temporarily reversed in a different task context, demand activation of a frontoposterior cortical network for updating episodic task-set information (Barceló *et al.* 2002, 2006).

In a recent study (Barceló & Knight, in press), we observed that dorsolateral prefrontal cortex is necessary for establishing the contextual meaning of novel events either as irrelevant distracters in an unpredictable context (i.e., pathway *sx-r0* in Fig. 1a), or as anticipatory cues for target and action selection in a predictable context (i.e., pathway *sx-r1* in Fig. 1a; Barceló & Knight 2000; in press). Unilateral prefrontal lesions disrupted novelty P3 activity in both hemispheres regardless of the predictive value or the hemifield of novel display (Fig. 1b). Moreover, the temporal contingency between predictive novels and targets was learned *only* when novels were displayed at the ipsilesional (good) visual hemifield of patients. In this condition, predictive novels elicited anomalous sustained early 50–200 ms negativities over the lesioned cortex (Fig. 1b; Ipsi Novels). The early timing of this anomalous negativity, onsetting before visual information could reach prefrontal cortex through geniculostriate pathways, suggested incoming signals from a prefrontotectal route that could not be adequately dealt with because of missing prefrontal task-set representations. The inability to learn the novel-target contingency when predictive novels were flashed contralesionally concurs with these patients' target neglect and other superordinate deficits in cognitive control (i.e., anosognosia). From an information theoretic approach to brain function, both exogenous (i.e., collicular) and endogenous (i.e., prefrontal) sources of information are necessary to compute the informational content of sensory signals (Fig. 1a). However, the meaning of human conscious experience seems to emerge from large-scale cortical dynamics, with the prefrontal cortex acting as the chief executive in the hierarchy of cognitive control (cf. Fuster 1997).

The hypthalamo-tectoperiaqueductal system: Unconscious underpinnings of conscious behaviour

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Abstract: The insight that, in terms of behaviour control, the mesodiencephalic system is superordinate to the cortex should have profound implications for behavioural sciences. Nevertheless, the thalamocortical system could still be deemed an "organ of consciousness" if we came to accept that consciousness is not central to purposeful behaviour, in accordance with instinct theory. Philosophically, Merker's concepts of basic consciousness and ego-centre warrant critical discussion.

I begin with a long quote from William James' *The Principles of Psychology*, which considers the nature of self-experience in relation to action and consciousness:

> If we divide all possible physiological acts into *adjustments* and *executions*, the nuclear self would be the adjustments collectively considered; and the less intimate, more shifting self, so far as it was active, would be the executions. But both adjustments and executions would obey the reflex type … The peculiarity of the adjustments would be that they are minimal reflexes … uninteresting except through their uses in furthering or inhibiting the presence of various things and actions before consciousness … These characters would naturally keep us from introspectively paying much attention to them in detail, whilst they would at the same time make us aware of them as a coherent group of processes strongly contrasted with all other things consciousness contained – even with the other constituents of the "Self," material, social, or spiritual, as might be the case … Everything arouses them; for objects which have no other effects will for a moment contract the brow and make the glottis close … These primary reactions … are the permanent core of turnings-towards and turnings-from, of yieldings and arrests, which naturally seem central and interior in comparison with the foreign matters, apropos to which they occur, … It would not be surprising, then, if we were to feel them as the birthplace of conclusions and the starting points of acts, or if they came to appear as … the "sanctuary within the citadel" of our personal life … it would follow that all that is experienced is, strictly considered, objective; that this Objective falls asunder into two contrasted parts, one realised as "Self," the other as "not-Self;" and that over and above these parts there is nothing save the fact that they are known, the fact of the stream of thought being there as the indispensable subjective condition of their being experienced at all. (James 1890, pp. 302–304)

Merker should be applauded for emphasising the evolutionary significance of the mesodiencephalic system – comprising hypothalamus, periaqueductal gray, and superior colliculus – and pointing out that the cerebral cortex is at the service of this system. The insight that more primitive upper-brainstem-based mechanisms occupy a superordinate position in the regulation of behaviour does not mean, however, that consciousness, too, is merely elaborated by the cortex. The superior colliculus implements a form of "analog reality simulation"; however, it seems unjustified to infer that such simulation in its interaction with action representations "constitutes a conscious mode of function" formed under the influence of "feelings reflecting momentary needs" (sect. 4.2, para. 5). Reality simulation biased by motivational variables and target selection may be crucially dependent upon mesodiencephalic structures indeed, but,

insofar as it becomes conscious (i.e., insofar as we can speak of *feelings* and the *experience* of an external world), it may still have to involve the thalamocortical system. Consistent with psychoanalysis, behaviour is primarily instinctive, and even social behaviour remains unconscious to a large extent. Consciousness starts to play a role when behavioural impulses arising in upper brainstem systems need to be delayed and modified – with reference to past experience – to adjust to complexities and variations in the interplay between multiple and conflicting goals and unpredictable opportunities and obstacles.

If the mesodiencephalic system centred on the superior colliculus were to provide "a connective interface between the brain's basic motivational systems and the orienting machinery" (sect. 4.2, para. 7) *as well as* the connectivity needed for consciousness, how can we understand aspects or sequences of goal-directed and motivated behaviour that are unconscious? Moreover, how are we to understand forms of consciousness that are relatively uncoupled from observable behaviour and clearly unrelated to sensory information being forwarded to the colliculus (dreams and hallucinations)? Conscious experience in dreaming and wakefulness is similar phenomenologically (Behrendt 2006) and accompanied by similar patterns of thalamocortical activity (Llinas & Pare 1991; Llinas & Ribary 1993), qualifying them as fundamentally equivalent states. In dreams and hallucinations, thalamic relay cells are less responsive to sensory stimulation while brainstem-based arousal mechanisms continue to activate thalamocortical circuits (Behrendt 2003). Here, conscious experience is uncoupled from sensory input representing the external world, and it seems unlikely that changes in thalamocortical activity elaborating the content of conscious experience in these states are paralleled by *corresponding* activity changes in the superior colliculus, in contrast to Merker's testable prediction, although the inferior colliculus was active during auditory hallucinations (Shergill et al. 2000).

Merker's hypothesis crucially depends on the notion that consciousness is "the 'medium' of any and all possible experience" (sect. 1, para. 3), and therefore that consciousness can be separated from the content of experience – that there can be consciousness without content. Indeed, he treats consciousness as a "functional utility" that is "independent of the level of sophistication at which the contents it integrates are defined" (sect. 1, para. 6); and it is only from this position that we can interpret Penfield and Jasper's (1954) findings as suggesting that "hemispherectomy does not deprive a patient of consciousness, but rather of certain forms of information, discriminative capacities, or abilities, but not of consciousness itself" (sect. 2, para. 3). This position may also misguide us to look for a "way in which this medium might be implemented neurally" (sect. 1, para. 4); and when pinning primary consciousness to "quite specific neural arrangements" one comes to the rather paradoxical conclusion that anencephalic children who "show responsiveness to their surroundings in the form of emotional or orienting reactions to environmental events" (sect. 5, para. 6) – such as sounds and "salient visual stimuli" – are conscious, whereas purposefully reacting invertebrates, such as the medusa, which lack such "specific structural arrangements" (sect. 1, para. 4) are not.

What is more problematic is that by *reducing* consciousness to "the kind of responsiveness to their surroundings that qualifies as conscious by the criteria of ordinary neurological examination" (sect. 5, para. 4), Merker ignores the subjective nature of conscious experience (Searle 1992, 1997). Signs of pleasure or excitement exhibited by anencephalic children are not necessarily indicative of conscious experience and can only impress the reductionist as "a weighty piece of evidence regarding their conscious status" (sect. 5, para. 7). They may be regarded more parsimoniously as automatic "molar" behaviour patterns represented in mesodiencephalic structures and activated by suitable stimuli. The fact that some patients with damage to the striate cortex can recognise or discriminate visual stimuli presented in their blind visual field in the absence of awareness (blindsight) (Weiskrantz 1996) illustrates that "environmental sensory information is related to bodily action (such as orienting)" (sect. 5, para. 10) *not* necessarily through the medium of a "primary consciousness." Decorticate animals orient to their surroundings and display molar behavioural reactions, suggesting indeed that these behaviours are dependent on structures in the mesodiencephalic region, but they too may do so without conscious awareness. Cortical blindness following destruction of posterior cortical visual areas can be restored by inactivation of the contralateral superior colliculus (Sprague effect); however, the restoration in the formally blind field is "limited essentially to the ability to orient to and approach the location of moving visual stimuli" (sect. 3.1, para. 1), so that we cannot be confident that the orienting behaviour now under control of the ipsilateral superior colliculus is conscious, that is, that we are dealing with a "partial restoration of vision" (sect. 3.1, para. 2) in the sense of a conscious function.

Merker appreciates that "what we confront in sensory consciousness is indeed a simulated (synthetic) world and body" (sect. 4.3, para. 5), concurring with philosophical idealism (Behrendt 2006). Problematic, however, is the notion of "egocenter" (sect. 4.3), which "we ourselves occupy when we are conscious" (sect. 4.3, para. 2) and which is thought to be located at the "origin of the coordinate system of the simulation space" (sect. 4.3, para. 2). Passivity phenomena in schizophrenia suggest that there is *no* "irreducible asymmetry . . . between perceiving subject and apprehended objects" (sect. 4.3, para. 2). More likely, basic sensorimotor self experience is a *derivative* of instinct-driven conscious behaviour: Tension reduction during approach to a desired goal – the yielding to an urge or impulse, often after overcoming conflicting drives – which accompanies all consciously guided behaviour and thinking, introduces an asymmetry between self and non-self into the unitary realm of subjective conscious experience (Behrendt 2004; 2005), which according to philosophical phenomenology and idealism is all that is available to us (see the quotation from James [1890] at the beginning). We are, in other words, not "central residents of that simulation" and as such "subject to ever shifting moods, feelings, urges, emotions, and impulses" (sect. 4.3, para. 6), but we ourselves are the product of these urges, emotions, and impulses (Behrendt 2004; 2005). The postulation of "an inherently 'perspectival,' viewpoint-based, relation to the contents of sensory consciousness" (sect. 4.3, para. 2) is unnecessary and *does not* accord with what Schopenhauer (1819/1958) meant when he stated that the subject as the *bearer of the world* is in itself unknowable – that the knowing and representing subject (the material underpinnings of the realm of conscious experience) cannot be found in the world that is experienced (Behrendt 2006).

Subcortical consciousness: Implications for fetal anesthesia and analgesia

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Abstract: In this commentary we discuss the possibility of subcortical consciousness and its implications for fetal anesthesia and analgesia. We review the neural development of structural and functional elements that may participate in conscious representation, with a particular focus on the experience of pain.

Is a cortex required for consciousness? If we adopt the view of Hameroff (2006) that consciousness in its most basic form may be considered "minimal awareness" without a requirement for memory, cognition, or organizational sophistication, then Merker makes a compelling argument that subcortical structures are both necessary and sufficient. In this context Merker discusses the ethical administration of anesthesia and analgesia to children with hydranencephaly, as well as neonates. In an era in which prenatal interventions are increasingly common, such ethical questions now apply to the developing fetus. If a fully mature cortex is not required for consciousness, at what point in development can the fetus potentially feel pain? Within Merker's paradigm, the possibility of fetal pain depends on the development of the structural and functional apparatus for subcortical processing. If we consider "pain" to be the coordinated, subjective experience of nociception, then "pain" may serve as a functional surrogate for consciousness. Analysis of the development of pain pathways may inform our understanding of the structural and temporal development of consciousness itself.

The first essential requirement for nociception and pain is the presence of sensory receptors, which develop first in the perioral area at around 7 weeks gestation. From 11 weeks, they develop in the rest of the face and in the palmar surfaces of the hands and soles of the feet. By 20 weeks, they are present throughout all of the skin and mucosal surfaces (Smith 1996). The nociceptive apparatus is initially involved in local reflex movements at the spinal cord level without supra-spinal integration. As these reflex responses become more complex, they subsequently involve the brainstem, through which other responses, such as increases in heart rate and blood pressure, are mediated. Such reflex responses to noxious stimuli have not been shown to involve the cortex and, thus, traditionally have not been thought to be available to conscious perception (Myers & Bulich 2005). Merker's article brings this into question.

Penfield and Jasper (1954), however, suggest that cortical structures are at least in some way required. The subcortical system – including the basal ganglia, medial thalamus, ventrolateral thalamus, substantia nigra, ventral tegmental area, superior colliculus, median raphe, and the midbrain and pontine reticular formation – does not function "by itself alone, independent of the cortex," but "by means of employment of various cortical areas" (Penfield & Jasper 1954, pp. 173–74; see target article, sect. 2, para. 7). Therefore, if integrative thalamic function is necessary for nociceptive perception (i.e., "pain") or any other higher-order sensory perception, it cannot be until the thalamocortical connections are formed and functional. The thalamus is first identified in a primitive form at day 22 or 23 post-conception. Its connections grow out in phases, initially only as far as the intermediate zone of the cerebral wall, collecting below the cortical plate. The neurons then advance further into the cerebral hemispheres, eventually becoming localized into their specific functional fields. The final thalamocortical connections are thought to be in place by around 26 weeks, although estimates differ (Royal College of Obstetricians and Gynecologists 1997). In fact, there are thought to be transient cholinergic neurons with functioning synapses connecting the thalamus and cortical plate from approximately 20 weeks (Kostovic & Rakic 1990). This point could be considered the absolute earliest time in gestation when a fetus could be aware of nociceptive stimuli.

The presence of electroencephalographic (EEG) activity would suggest a degree of *functional* maturity, in addition to *structural* maturity, of neural systems mediating consciousness. While sporadic electrical activity has been detected in the fetal brain as early as 43 days gestation (Holzman & Hickey 2001), more coordinated electrical activity (in the form of intermittent bursts) has been shown to be present in the brainstem from 12 weeks, and the cerebral hemispheres at 20 weeks (Myers & Bulich 2005). Before 25 weeks, the electrical activity on EEG recordings is discontinuous, with periods of inactivity lasting up to 8 minutes and bursts of activity of only 20 seconds (accounting for only 2% of the total time). From 25 to 29 weeks, the periods of activity increase, such that by 30 weeks, although EEG activity is still not continuous, distinct patterns of wakefulness and sleep can be recognized as the precursors of adult patterns. These are not initially concordant with behavioral state; over the next few weeks, however, the degree of concordance improves (Clancy 1998). By 34 weeks, electrical activity is seen 80% of the time; from 34 to 37 weeks, sleep/wake cycles become more defined (Myers & Bulich 2005).

Although current studies cannot provide direct evidence of fetal consciousness, they do suggest that the required neural processing architecture may be in place and functional. If we are to accept that by approximately 20 weeks the requisite neural substrate of consciousness (e.g., the thalamus and associated subcortical structures) and its proper connections are in place and accompanied by a coordinating EEG rhythm (even if only intermittently), what can we say about the beginning moments of fetal consciousness? Again, it would seem that we can conclude that consciousness is at least *possible* from this point forward in fetal development. If a more stringent threshold for continuous EEG activity is required, then it would appear that by 30 weeks gestation, when patterns consistent with wakefulness and sleep may be discriminated, consciousness is at least *possible*.

If we accept that a subcortical consciousness is possible by 20 weeks (or, more conservatively, 30 weeks), then it also would appear possible that fetuses could experience something approximating "pain." Surely, the complex behavioral responses seen in ventilated neonates have the external appearance of pain, but because we currently have no metric with which to make such a determination, we cannot know this with any certainty. The mere *possibility* of consciousness and an experience of pain – however rudimentary – would mandate a provision of appropriate anesthesia and analgesia. Merker would appear to agree, as the evidence surveyed in his article gives no support for consciousness as an exclusively cortical function. Rather, he implies that subcortical structures may be necessary and sufficient to generate consciousness and, therefore, a rudimentary experience of pain. As such, his challenge to the medical community has significant ramifications for medical ethics, as well as the provision of fetal anesthesia and analgesia.

Consciousness without a cortex, but what kind of consciousness is this?

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Abstract: Merker suggests that the thalamocortical system is not an essential system for consciousness, but, instead, that the midbrain reticular system is responsible for consciousness. Indeed, the latter is a crucial system for consciousness, when consciousness is regarded as the waking state. However, when consciousness is regarded as phenomenal consciousness, for which experience and perception are essential elements, the thalamocortical system seems to be indispensable.

Structures in the upper brainstem mediate consciousness by activation and arousal of the entire thalamocortical system, thus producing the waking state. When the mesencephalic reticular formation becomes active, the activity in the thalamocortical loops rise, together with an opening of the sensory channels. A stream of information from the outside world flows to the higher brain centers and is perceived. Numerous neuronal systems start to process and integrate this information and the

activity of myriads of neurons firing in the tonic mode is expressed in consciousness, a sort of neural orchestra. It is a common assumption that the neuronal basis of consciousness results from the interactive processes between the brain stem reticular formation and the thalamocortical system (Coenen 1998).

When the activity of the mesencephalic reticular formation drops under a critical level, an inhibitory system becomes active and starts to inhibit the thalamocortical neurons. Then, these neurons are tied together by the inhibitory interneurons and discharge irregularly in a burst-pause mode. Slow wave sleep is the result. Because of "thalamic gating," sensory information is largely blocked during sleep and information processing is at a low level. Perceptive processes are minimal and consciousness is also at a low level (Coenen 1998). The interaction between the midbrain reticular formation, the nonspecific diencephalic nuclei and the thalamocortical system seems to control the high consciousness during sleep and the low consciousness during slow-wave sleep.

Absence epilepsy is a form of non-convulsive epilepsy, occurring in children as well as in animals. The basic characteristic of this type of epilepsy is the reduction in responsiveness and consciousness, associated with spike-wave discharges in the electroencephalogram. The "centrencephalic" theory suggests that these aberrant brain discharges originate from a deep-seated intrathalamic pacemaker extending to the midbrain reticular formation (Penfield & Jasper 1954), whereas recent research points towards a prominent role for the cortex in this process (Meeren et al. 2005). Absence seizures are characterized by lapses in consciousness and a lack of response towards external stimuli. Absence seizures share many similarities with slow-wave sleep (Coenen 1999). Already mentioned is the reduction in consciousness and the unresponsiveness to sensory stimulation. Despite the reduction in responsiveness, both states can be terminated by strong stimuli. Another correspondence is that unconscious stimulus evaluation still seems possible. Relevant stimuli can terminate both slow-wave sleep and absence attacks more easily than neutral stimuli. This also shows that some consciousness is still present during both states. Presumably, all phenomena can be related to the underlying neuronal mechanisms. In both the sleep state and the absence state, neurons are firing in the "burst firing" mode. A difference is the regular and spiky character of the spike-wave discharges, which could be a result of the even stronger burst firing mode during absences (Coenen 1995). The midbrain reticular formation is inhibited in both states, which implies a reduction in consciousness. A firm conclusion is inevitable: an active midbrain reticular system is a necessary condition for consciousness. This agrees well with the conclusion of Merker.

But what is the role of the thalamocortical system in consciousness and can consciousness exist without the thalamocortical system? These are the intriguing questions faced by Merker. He concludes that the thalamocortical system cannot alone be regarded as "the organ of consciousness"; instead, it is the "centrencephalic system" or midbrain reticular system that seems to play main fiddle in consciousness. Or in Merker's own words "brainstem mechanisms are integral to the constitution of the conscious state" and "neural mechanisms of conscious function cannot be confined to the thalamocortical complex alone" (target article, Abstract). One of the central questions, however, is what Merker means by consciousness. Despite several explanations, the meaning of this hard to define and difficult concept is not clear to all. Zeman (2001), in his extensive review, distinguishes from among the eight meanings of consciousness, two principal meanings. The first is, "consciousness as the waking state" and the second is, "consciousness as experience." Consciousness in the first sense is the behavioral expression of the waking state. Being conscious in that sense is synonymous to being alert and awake. The second sense of consciousness, however, refers to becoming aware of something and to experience something, which is often called "phenomenal consciousness" (Block 1995). The essence of phenomenal consciousness is inextricably bound up with experience and perception, for which the thalamocortical system is mainly responsible. Philosophers often use the term "qualia" to highlight the subjective dimensions of experience and perception. Consciousness in the first meaning (consciousness as the waking state), is in this view a necessary condition for consciousness in the second sense (consciousness as experience or phenomenal consciousness).

Going back to the meanings of consciousness in the interaction of the midbrain reticular and thalamocortical systems, the following picture emerges. The midbrain reticular system takes care of wakefulness and arousal, it brings the thalamocortical system into a state conducive for experience and perception, leading to the processing and integration of information, and thus to consciousness in the second sense. The midbrain reticular system acts as the medium for phenomenal consciousness. It forms the engine of the car, while the vehicle itself (the thalamocortical system) is necessary for driving the car. Hence, I agree with Merker's view that consciousness can exist without a cortex, and at the same time I disagree with Merker's view that consciousness can exist without a cortex. It depends on the type of consciousness. Waking consciousness is possible with the midbrain reticular system alone, but phenomenal consciousness is not possible without the thalamocortical system. Two intact systems are necessary for consciousness: the midbrain system for waking and vigilance (the engine), and the thalamocortical system for perception and experience (the vehicle). That children without a cortex may experience some phenomenal consciousness, might be explained by the fact that parts of the extensive thalamocortical system are still functional.

Do multiple cortical–subcortical interactions support different aspects of consciousness?

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Abstract: Merker's core idea, that the experience of being conscious reflects the interactions of actions, targets, and motivations in the upper brainstem, with cortex providing the content of the conscious experience, merits serious consideration. However, we have two areas of concern: first, that his definition of consciousness is so broad that it is difficult to find any organisms with a brain that could be non-conscious; second, that the focus on one cortical–subcortical system neglects other systems (e.g., basal forebrain and brainstem cholinergic systems and their cortical and thalamic target areas) which may be of at least equal significance.

Bjorn Merker has to be admired for entering the debate on the question of the location of consciousness with the bold assertion that the cortex is not essential. His core proposal, that the experience of being conscious reflects the interactions of systems supporting actions, targets, and motivations in the upper brainstem, with cortex providing the content of the conscious experience, is novel. It seems highly likely that upper brainstem systems projecting to the superior colliculus are important components of integrative networks that support consciousness in mammals. However, we argue that they are neither quite so critical nor as unique as he suggests.

One area of concern is that Merker's use of the term "consciousness" is too broad to allow a clear focus on specific brain areas. The definition of consciousness as being a "state or activity

that is characterized by sensation, emotion, volition, or thought" (sect. 1) could include, in its most basic sensation form, receiving, processing, and responding to any environmental signal or information. Such a definition is applicable not only to mammals, but also to most animals with a cerebrum, no matter how different from humans (Edelman et al. 2005). Innumerable nonliving mechanisms might also fit the bill.

Such a very broad use of the term "consciousness" both undermines Merker's use of mammalian evolutionary homologies to support his localisation in the brainstem and weakens the importance of his evidence from children born without a cortex. We entirely agree on the need to see each child's individual capabilities and not draw conclusions from diagnostic labels. However, his scientific case would be strengthened if he could show that there was no relationship between variations in consciousness and residual amounts of cortex. The more restricted use of the term "consciousness" that he later seems to favour, involving subjective awareness (more analogous to self awareness in Morin's 2006 taxonomy), may localise to a smaller range of neurobiological structures.

Merker uses evidence of consciousness in the absence of cortex in rats and children to argue that brainstem structures are of primary importance to the conscious experience. However, this data is also consistent with consciousness being the product of a resilient distributed neural network (or network of networks). Arguing against a single consciousness system, damage in restricted brain areas – for example, from strokes (Goldstein & Simel 2005), provided arousal is not grossly impaired – rarely abolishes consciousness entirely, though it may well limit the areas to which it can be applied. Thus, unilateral spatial neglect (sect. 3.1) suggests that consciousness can be fractionated, at least in space, and perhaps in modality.

In order for upper brainstem systems to be especially relevant within these networks, Merker would have to show that lesions within the superior colliculus, for example, have profound effects on consciousness. However, collicular lesions generally impair orienting rather than consciousness (sect. 4.5, see also Burnett et al. 2004), and the gross disturbances in consciousness common after brainstem strokes are due to the disruption of the ascending cholinergic and other projections, which we discuss further on.

The neuropathology of diseases that disturb consciousness can provide important insights. Parkinson's disease (PD) with its relatively specific nigral dopaminergic loss, which leads to gross basal ganglia dysfunction, can test the role of the basal ganglia input to the superior colliculus within his model. Pathology in this system does lead to eye-blink abnormalities (Basso et al. 1996) and, consistent with Merker's hypothesis, visual hallucinations (a disorder of the content of consciousness), and disturbed dream content and behaviour occur in PD, as well (Olson et al. 2000; Onofrj et al. 2006). (We consider that dreaming is a normal state of altered consciousness). However, such disorders of consciousness are even more closely associated with the related disorder, Dementia with Lewy Bodies (DLB; Boeve et al. 2004; Collerton et al. 2005). Additionally, the fluctuating basal ganglia function in PD leads primarily to fluctuating motor symptoms (Denny & Behari 1999); not to the fluctuations in consciousness that are seen in DLB (Bradshaw et al. 2004; Walker et al. 2000). Pathology in DLB extends far beyond Merker's brainstem system, and includes clinically relevant disturbances in cholinergic systems (Fujishiro et al. 2006; Lippa et al. 1999; Perry et al. 1993; Tiraboschi et al. 2002; Ziabreva et al. 2006), which may also be important in conscious experience.

The basal forebrain cholinergic system, with its multiple projections to GABA and glutamate neuronal networks in the cortex and thalamic regions, and its role in both tonic and phasic activation via specific nicotinic and muscarinic receptor subtypes is, in conjunction with cholinergic projections from the brainstem to key areas such as thalamus and substantia nigra, a candidate integrative mechanism underpinning the emergence of consciousness from unconscious mental activity (Perry et al. 1999).

Dreaming and anaesthesia also support a central role for the interaction of cholinergic projections and cortical target areas in modulating conscious awareness. Between sleep (non-REM and REM) and waking, alterations in basal forebrain cholinergic activity correlate with concomitant changes in consciousness, to a greater extent than in monoaminergic and other systems (Perry & Piggott 2000). Among drug-induced changes in consciousness, mechanisms of general anaesthetic-induced disruption of the effective connectivity and integrative processes required for consciousness is considered likely to provide insights into neural correlates of consciousness (Mashour 2006). It is well established that neuronal nicotinic acetylcholine receptors are particularly sensitive to inhalational anaesthetics (Rada et al. 2003). For example, isoflurane, sevoflurane, and halothane potently block the $\alpha4\beta2$ nicotinic subtype (Yamashita et al. 2005). Alterations in the same nicotinic receptor subtype in temporal cortex and thalamus are related to disturbances in consciousness in DLB (Ballard et al. 2002; Ray et al. 2004; Pimlott et al. 2006).

We have argued that brainstem and basal forebrain cholinergic projections to the ventral visual stream, lateral frontal cortex, and connecting structures (Collerton et al. 2005, Fig. 7) form a distributed system for conscious visual processing (Collerton et al. 2005, Fig. 3). Dysfunctional conscious awareness – visual hallucinations – can result from subcortical cholinergic dysfunction incorrectly modulating the balance between top-down and bottom-up processing within the cortex. The disturbance, in this case, therefore lies within a cortical–subcortical system distinct from that described by Merker.

Consistent with a cholinergic component of consciousness and the suggestion that Merker's system is one among many supporting consciousness, not only the superior colliculus but other key "hub"/central station areas in the brain that collect a multiplicity of afferents from and distribute efferents to essential areas such as brainstem, thalamus, or cortex (e.g., interpeduncular nucleus, many thalamic nuclei, in particular the lateral geniculate, the substantia nigra pars compacta, and the septum, subiculum, and parahippocampal gyrus) are relatively very high in nicotinic receptors; especially $\alpha4\beta2$ (Han et al. 2003; Perry & Kellar 1995; Perry et al. 1993; 1995; Spurden et al. 1997) which facilitates GABA inhibition (Endo et al. 2005).

We therefore conclude that Merker has not quite made his case that the cortex is inessential in conscious experience, but that he has very helpfully provided a new focus on the need to incorporate subcortical mechanisms as well.

Pain, cortex, and consciousness

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Abstract: Painful stimuli evoke functional activations in the cortex, but electrical stimulation of these areas does not evoke pain sensation, nor does widespread epileptic discharge. Likewise, cortical lesions do not eliminate pain sensation. Although the cortex may contribute to pain modulation, the planning of escape responses, and learning, the network activity that constitutes the actual experience of pain probably occurs subcortically.

Pain is a sensory and emotional quality experienced by a conscious brain. There has never been much doubt that the pathways leading to pain perception, like all other conscious

experience, end in the cerebral cortex. However, closer consideration of this dogma raises some perplexing questions.

Microelectrode recordings in animals, and noninvasive functional imaging in humans, show excitations in many brain areas following pain-provoking stimulation of the skin and internal organs (Peyron et al. 2000). These include structures long known as key parts of the somatosensory system, such as the thalamic nuclei VPL-VPM and S1 and S2 cortex, as well as areas not classically thought of as somatosensory processors, such as the cerebellar cortex and the corpus striatum. Curiously, the most robust and reliable cortical activations occur not in S1 and S2 but in limbic cortical areas, including the anterior cingulate cortex (ACC) and the posterior insular cortex. Noxious stimulation of different organs – skin versus viscera, for example – reveal different if overlapping patterns of cortical activation, appropriate to the different "feels" evoked. Moreover, these cortical activations, particularly in ACC, track reported pain unpleasantness and not the intensity of the applied stimulus when the two are dissociated by manipulations such as placebo and hypnotic suggestion (Rainville et al. 1997; Strigo et al. 2003).

All of these observations are as expected of a cortical pain analyzer. However, other observations are not as expected. The most important is that direct electrical stimulation of the cortical convexity, including areas activated by painful stimuli, almost never evokes a report of pain in awake patients (Libet 1973; Penfield & Rasmussen 1955). Likewise, for transcranial magnetic stimulation (TMS). This contrasts with stimulation of cortical areas associated with vision, hearing, smell, and (non-painful) touch, which readily arouses the corresponding percepts. It may be argued that the structures relevant for pain sensation are buried in the mid-sagittal (ACC) or Sylvian sulci (insula) and are hard to access by surface stimulation. A related explanation is that unlike the other senses, multiple cortical areas must be activated simultaneously to evoke a sensation of pain. However, in epileptic seizures cortical discharge is frequently widespread and includes, indeed often favors, these buried limbic cortices. Nonetheless, it is very rare for epilepsy to include auras that are painful (Nair et al. 2001). A recent report of pain evoked in a small number of epileptic patients by depth electrodes on the insular cortex is a potential exception (Mazzola et al. 2006). However, it has been shown that direct stimulation of the meninges and blood vessels that overly the insular cortex evokes pain sensation (Pereira et al. 2005). These structures have rich nociceptive innervation from the trigeminal ganglion. Thus, the claim that pain is evoked by insular stimulation using depth electrodes may be confounded by inadvertent simultaneous stimulation of local non-neural tissues. That is, the reports of pain on insular stimulation may not actually be due to activation of the insular cortex. Note that in contrast to the cortex, pain is readily evoked by focal (microelectrode) stimulation in certain areas of the thalamus and brainstem (Dostrovsky 2000).

Another retort sometimes given in response to the question of why cortical stimulation is so rarely painful is that pain is complex and is multiply represented in the cerebral hemispheres. As a consequence, unlike vision, hearing, smell, and touch, to evoke pain by cortical stimulation would require precisely patterned stimulation, simultaneously, at many locations. This condition is neither met by Penfield-type stimulation experiments, nor is it found in natural seizures. However, if evoking a pain percept requires such precise, complex, and necessarily fragile patterning of activity, then disruption of the pain network at any of numerous loci ought to eliminate the ability of natural stimuli to evoke pain sensation. In fact, focal lesions in cortical areas active during pain, and even massive cortical lesions, do not produce analgesia. On the contrary, cortical strokes are often followed by chronic neuropathic post-stroke pain (Boivie et al. 1989). Lesions in cortical areas thought to subserve vision, hearing, smell, and touch do not behave in this way. Patients with large lesions in the primary visual cortex, for example, are perceptually blind, although they may have some residual visually guided function. Why, then, do large lesions in the somatosensory areas of the cortex, or any cortical region for that matter, not render people "blind" to noxious stimuli, that is, make them pain-free?

These observations demand that one at least consider the possibility that the neural computations that generate pain experience play out subcortically rather than in the cerebral cortex. Certainly, focal electrical stimulation at many subcortical sites, from the spinal cord to the thalamus, is able to provoke pain sensation. Patients with lesions in the right parietal cortex sometimes show sensory neglect, denying that a body part (arm, leg) belongs to them. However, noxious stimulation of the denied limb evokes normal wincing, autonomic responses, and withdrawal. Pain is experienced and acknowledged, but is missing a location in the body schema. Finally, people with massive cortical lesions that qualify them for the diagnosis "persistent vegetative state," anencephalic children, and decorticated animals, all show organized, adaptive "nociflexive" behavior in response to noxious stimuli. True, such behavior, in itself, does not prove that the noxious stimulus has been experienced as pain by a conscious brain. It only proves that the noxious stimulus has been registered and basic adaptive motor sequences have been generated in response. Nonetheless, in light of the possibility that pain perception does not require cortical function, a decision to end the life of a vegetative patient ought to be carried out painlessly using a fast-acting agent, rather than by withholding life support and condemning the patient to a month or more of starvation.

Corticothalamic necessity, qualia, and consciousness

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Abstract: The centrencephalic theory of consciousness cannot yet account for some evidence from both brain damaged and normally functioning humans that strongly implicates thalamocortical activity as essential for consciousness. Moreover, the behavioral indexes used by Merker to implicate consciousness need more development, as, besides being somewhat vague, they lead to some apparent contradictions in the attribution of consciousness.

Merker has done an excellent job of bringing the centrencephalic proposal of Penfield and Jasper up to date. We wish to sharpen the contrast between Merker's updated proposal and the proposal that the thalamocortical system, instead, constitutes the fundamental neural substrate of consciousness. The possibilities regarding the respective roles of the mesodiencephalic system described by Merker and thalamocortical system are three: either one, or the other, or both are necessary and sufficient for the existence of the conscious state. In this commentary we adumbrate evidence that the thalamocortical system is necessary, if not sufficient, for conscious awareness as experienced by humans. These data are difficult to account for in the mesodiencephalic proposal, as are, in turn, some data discussed by Merker for the thalamocortical proposal. An unsatisfying but reasonable conclusion is that both systems play crucial roles in the generation of the conscious state.

Merker argues for the existence of consciousness in humans without a cerebral cortex, at least partly, on the basis of the behavior of hydranencephalic children who "are not only awake and often alert, but show responsiveness to their surroundings in the form of emotional or orienting reactions to environmental

events [. . .], . . . express pleasure by smiling and laughter, and aversion by 'fussing,' . . . and show preferences for certain situations and stimuli over others" (target article, sect. 5, para. 6). Earlier, the cubomedusa is given as an example of a species that cannot possess consciousness because of its simplistic, non-cephalized, nervous system architecture. Cubomedusa, like other even simpler organisms such as *C. elegans* (e.g., Rankin 2002), should display responsiveness to external stimuli, approach and aversion, and conditioned preferences for certain stimuli and situations (although many of these experiments appear not to have been done for cubomedusa). It *does* display coordinated mating and hunting behavior as well as avoidance of particular obstacles (e.g., Coates 2003). If the cubomedusa can display such behaviors without consciousness, then so, perhaps, can hydranencephalic humans.

Conversely, it has been proposed that the nerve ring that connects ganglia near the eyes and swimming effectors in the cubomedusa serves to integrate visual information for action in the most effective way for a radially symmetric organism (Coates 2003). If behaviors such as those listed earlier indicate the capacity for conscious experience, and given its nerve ring mechanism to provide neural integration, it seems possible that even the cubomedusa experiences its visual environment in a crude and primitive way. Thus, cephalization might not be necessary for conscious experience.

Neither of these conclusions is particularly palatable, although each is reasonable and potentially correct. The difficulty in finding useful behavioral indicators underscores the importance of centering our inquest into the neural correlates of consciousness where we can be most certain about whether consciousness is present, namely, in neurologically normal adult humans or in human subjects in which brain damage has resulted in a reportable loss of consciousness. Disorders of awareness reveal some inconsistencies with the mesodiencephalic theory of consciousness that need to be accounted for. Consider, for example, cortical blindness, or "blindsight," which is a loss of visual awareness induced by damage to the striate cortex. Residual *nonconscious* visual functions in blindsight have been attributed to the superior colliculus and its inputs to the cortex (Leh et al. 2006). Hence, in otherwise normally conscious humans, it seems that the visual and other information that is integrated in the superior colliculus *is not* consciously available. The Sprague effect does not resolve this issue, because what is recovered are subcortically mediated orienting responses similar to those demonstrated in blindsight. Thus, blindsight and similar pathologies (e.g., cortical deafness) constitute evidence for an apparent reliance of conscious experience on processing in the corticothalamic system.

A central tenet of the midbrain theory of consciousness is that, within the midbrain, a "winner take all" system exists, thereby accounting for the dynamic and integrated/unified stream of consciousness, furnished with the most salient perceptual and motor information. One problem with this idea is that the neural representations in the midbrain network do not possess the detail characteristic of human experience. Clear examples can be taken from the qualia of vision. Only in the cortex do representations possess sufficient definition in terms of form, motion, color, and spatial resolution to account for human qualia. Indeed, the complexity and integration inherent to processing in the thalamocortical system has been proposed to be essential for consciousness (Tononi 2004; Tononi & Edelman 1998). Descending afferents to the superior colliculus result in representations in which sufficient information reduction has occurred to make them inconsistent with the fine grain of our experience. Although midbrain systems could be sufficient for a crude and primitive form of consciousness, it is unclear how this system could account for the everyday consciousness of adult humans. Does the corticothalamic system "take over" as the seat of consciousness in normal adults? Does the seat of consciousness now extend to a larger section of the brain? Are the various representation levels overlaid upon one another, and only the most detailed level experienced, as proposed by Brown (1988)? Here the Sprague effect in relation to blindsight should be reconsidered, as what is *not* recovered are functions requiring the more detailed representations characteristic of our qualia, such as pattern recognition (Loop & Sherman 1977).

Moreover, as much cortical activity is not experienced, there also must be a "winner take all" network in the cortex. Transient large-scale networks of synchronous neuronal oscillations, proposed as being a mechanism that underlies feature binding in sensory awareness (Engel & Singer 2001), could also operate to select a subset of cortical activity for integration into a conscious representation (Varela et al. 2001). Such a network could be responsible for excluding V1 activity, for example, from direct experience (e.g., Rees et al. 2002). Furthermore, disturbances of the thalamocortical rhythms characteristic of conscious CNS (central nervous system) states lead to the abolition or alteration of consciousness, as seen in coma, general anesthetics, schizophrenia, and epilepsy (Steriade et al. 1990). Such data need to be accounted for if midbrain structures are to supplant, or to join, the corticothalamic system as the primary candidates for the biological substrate of consciousness.

Consciousness without corticocentrism: Beating an evolutionary path

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Abstract: Merker's approach allows the formulation of an evolutionary view of consciousness that abandons a dependence on structural homology – in this case, the presence of a cerebral cortex – in favor of functional concordance. In contrast to Merker, though, I maintain that the emergence of complex, dynamic interactions, such as those which occur between thalamus and cortex, was central to the appearance of consciousness.

In the target article, Merker challenges the pervasive view of the cerebral cortex as necessary for consciousness, and in doing so, beats a path towards a view of consciousness that makes sense from an evolutionary perspective. Merker's arguments are grounded primarily in detailed anatomical and physiological observations, as well as clinical studies and first-hand observations of anencephalic children, and there is a strain in his perspective that is deeply consonant with a modern evolutionary view of nervous system form and function. But he resists the notion that complex inter-areal dynamics in the nervous system were a necessary basis for incipient consciousness. In contrast, I maintain that complex dynamic interactions – such as, but not limited to, those arising in thalamocortical circuitry – were central to the emergence of the conscious process.

Like Merker, I believe that consciousness may not be contingent upon the particular anatomy of the cerebral cortex. The probability that some birds are conscious (see Butler & Cotterill 2006; Edelman et al. 2005) suggests that differently organized brain nuclei, with perhaps less well-defined lamina than mammalian cortex, are up to the task of sensory integration and interaction with thalamic nuclei, thereby yielding conscious states (notwithstanding the suggestion that the avian "wulst" is a cortical homolog, a notion that remains controversial; see Karten 1997). If consciousness emerged independently in the avian and mammalian lines (or in their reptilian, or stem amniote, precursors), it involved the elaboration of quite different structures serving identical functions. The centrencephalic system, *sensu stricto*, may not be necessary for conscious states. Moreover, invertebrate species, such as the cephalopod molluscs, with nervous

systems that are radically different in their organization than those of vertebrates, may well have some form of primary consciousness (Mather, in press). Although this idea remains woefully untested, it nevertheless seems clear that neural structures with different evolutionary histories and developmental trajectories may subserve similar functions, including the dynamic interactions underlying conscious states.

In general, biological structures and their particular functions do not emerge entirely *de novo* in the course of evolution. Rather, natural selection shapes, or retrofits, what is already on hand. Hence, although the appearance of a cortical mantle certainly enriched the contents of consciousness, it did not necessarily mark the emergence of incipient consciousness. In a given lineage, a certain function may predate the appearance of a structure which, in members of an extant species, has come to be associated with it. The new, or modified, structure may either have become part of a preexisting "circuit" serving this ancient function or simply co-opted the function entirely. Moreover, structural and functional convergences are not at all rare in the evolutionary histories of complex animals. Given what we can surmise from broadly comparative anatomical studies of present-day species, this seems to have been particularly true during the evolution of the nervous system and its associated sensory modalities. The oft-cited compound eye, which probably appeared a number of times independently in different evolutionary lineages (Oakley & Cunningham 2002; but see Gehring 2005), is an example of the latter.[1] The apparent convergent architectures (i.e., laminar structure) and physiologies (i.e., binocular vision) of the so-called avian wulst and mammalian neocortex (Medina & Reiner, 2004; Reiner et al. 2005) may be an example of the former. Surveying evidence from anatomical, physiological, and behavioral studies, my colleagues and I make precisely this argument in a recent paper (Edelman et al. 2005).

Merker's resurrection and substantive revitalization of Penfield and Jasper's (1954) "centrencephalic" hypothesis provides a novel anti-corticocentric view of consciousness. However, I disagree with his premise that elaboration of complex functional circuitry was not critical for the emergence of consciousness. The centrencephalic system appears to be the site of quite complex dynamic interactions between ascending (or attentional) systems, a relay locus, and integration centers. In two recent papers (Edelman et al. 2005; Seth et al. 2005), my colleagues and I suggest that a *sine qua non* of mammalian consciousness may be the dynamic interaction between thalamus and cortex, an idea first expressed by Edelman and Tononi (2000) in their "dynamic core" hypothesis nearly a decade ago. But, I will allow that, although reentrant thalamocortical loops may be the functional core of mammalian consciousness, theoretically neither cortex and thalamus, nor their underlying architectures, are necessary for conscious states. What consciousness requires, it seems, are richly and reentrantly connected structures that support essentially *the same functional interactions* as thalamus and cortex.

In making the case for consciousness in anencephalic children, Merker cites one published account documenting the assessment of four hydranencephalic children in which the authors conclude that all four children are conscious by the criteria of a standard neurological examination (Shewmon et al. 1999).[2] He also reports his first-hand impressions of the behavior of anencephalic children, as well as observations gleaned from the reports of parents of anencephalic children. Of these observations, perhaps most intriguing are reports that these children have seizures of absence epilepsy. In the case of anencephalic children, though, it is difficult to determine whether these individuals are conscious. Apart from limited behavioral means (obviously, no accurate verbal report is possible), there is little that can be done to test for conscious states. Collectively, Merker's accounts lack the weight of evidence. His anatomical sketch of the connectivity between midbrain structures, including the hypothalamus, periaqueductal gray, and superior colliculus, might suggest a neural substrate fully capable of complex integration of action and motivation, and thus generation of a detailed internal "world" map. Moreover, the absence of vast radial migrations of cortical progenitor cells during neurogenesis (as must be the case in anencephalic embryos) might allow further elaboration of otherwise deeper, subcortical, structures. But these prospects remain unverified and little explored.

The evolutionary implications of conscious states in animals that lack a cerebral cortex are ripe for exploration. Merker has made an intriguing foray into this realm, but much territory remains uncharted; an exciting prospect indeed.

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NOTES

1. Interestingly, certain regulatory genes that are important in the specification and patterning of structures such as eyes and, indeed, large portions of nervous systems and whole body plans, are widely conserved across invertebrate and vertebrate taxa. This insight, which emerged shortly after the discovery of the first homeotic, or hox, genes, is all the more tantalizing because the same hox genes expressed in representatives of disparate taxa have often been found to induce tissues of quite different embryonic origins to form functionally homologous structures during development (Carroll et al. 2001).

2. According to Merker, this is "the only published account based upon an assessment ... under near optimal conditions" (target article, sect. 5, para. 4).

Roles of allocortex and centrencephalon in intentionality and consciousness

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Abstract: "Decortication" does not distinguish between removing all cerebral cortex, including three-layered allocortex or just six-layered neocortex. Functional decortication, by spreading depression, reversibly suppresses only neocortex, leaving minimal intentionality. Removal of all forebrain structures except a hypothalamic "island" blocks all intentional behaviors, leaving only tropisms. To what extent do Merker's examples retain allocortex, and how might such residues affect his interpretations?

In considering the nature and functions of cerebral cortex, particularly as distinct from cerebellar cortex, it is useful to distinguish between two main categories. Three-layered allocortex (Mountcastle 1974, p. 232) is common to all vertebrate brains. It includes archicortex (hippocampus), paleocortex (prepyriform and periamygdaloid cortices), and the laminated neuropil of the olfactory bulb, though inclusion of the latter as "cortex" is controversial (Braitenberg and & Schüz 1998). Six-layered neocortex is found only in mammals, with transitional forms in marsupials; its well-known variants are distinguished by input-output connections and cytoarchitectures (e.g., Brodmann 1909).

A method for chemical decortication (Bures et al. 1974) relies on inducing the *spreading depression of Leão* to inactivate the cortex in each cerebral hemisphere. Under surgical anesthesia the scalp of the subject, usually a rat, is incised and reflected, and two small burr holes are made through the calvarium. The skin is closed loosely, and the animal is nursed to recover from the anesthetic. Then the skin is momentarily reflected, and a cotton pledget soaked in concentrated potassium chloride is placed over each burr hole. Within a minute or two the potassium

induces intense neuronal spiking that releases sufficient potassium ions into the intracortical extracellular space to precipitate a chain reaction that spreads in mm/minute over the entire neocortex in each hemisphere – but not beyond across the entorhinal fissure into the allocortices. The functional decortication lasts several hours and is fully reversible. Bures demonstrated "neodecortication" for me in Prague; on casual inspection I saw surprisingly little difference in the rat's behavior before, during, and after the process.

Phylogenetic evidence for the functions of allocortex stems from analyses of the brains and behaviors of simpler vertebrates, particularly the salamander (Roth 1987) – a neotenic amphibian that C. Judson Herrick (1948) regarded as the closest living descendent of the putative vertebrate ancestor. The three main parts of its forebrain are sensory (predominantly olfactory bulb with anterior olfactory nuclei), motor (pyriform cortex with paleostriatum), and associational (primordial hippocampus with septoamygdaloid nuclei). These components comprising the bulk of the primitive forebrain constitute the limbic system, which suffices to elaborate the goal-directed behaviors on which all vertebrates rely for survival.

The functions of these allocortical parts persist in mammals: most obviously, in support of olfaction, spatial orientation using the "cognitive map" (Jacobs 1994; O'Keefe & Nadel 1978), and temporal orientation in constructing a life history through learning dependent on short-term memory. These integrative processes are essential for intentional action into the world, because even the simplest search for food or shelter requires that an animal coordinate its position in the world and track its trajectory toward its target.

Selective partial removal of allocortex has profound effects on intentional behaviors. The bulbectomized rat provides the best biological model for intractable clinical depression (Jesberger & Richardson 1985; van Riezen & Leonard 1990). Damage to the mesial temporal lobes, which contain substantial parts but not all of the limbic system, results in severe loss of spatial and temporal orientation, compromising but not abolishing intentional behaviors or, apparently, consciousness. In contrast, bilateral destruction of selected areas of neocortex results in catastrophic but delimited losses in sensory and motor functions, including "social blindness" from frontal lobe damage, but not in loss of consciousness. I agree with Merker that the adaptiveness and flexibility of intent, the fullness of life-long memory in the unity of consciousness, and the cognitive contents of consciousness are elaborated by neocortex, but argue further that these three aspects are integrated predominantly in the allocortical limbic system (Freeman 2006), more than in Wilder Penfield's "centrencephalon integrating system" (Penfield & Jasper 1954).

On the one hand, the effects on behavior of full decortication have been studied in great detail for well over a century, beginning with the celebrated study of Friedrich Leopold Goltz (1892) that reportedly stunned his audience. The crucial work of postmortem verification of the extent of tissue removal was entrusted to an independent investigator at the beginning of his illustrious career, Sir Charles Sherrington. I have not seen Sherrington's report to the neurological congress in which Goltz reported his observations, so my question remains unanswered: Did Goltz surgically remove ("decorticate") parts or all of the allocortex or only neocortex, as in functional decortication? On the other hand, the removal of all cortex and striatum, leaving a hypothalamic "island" that is adequate for neurohumoral control (Bard & Rioch 1937) but not temperature regulation (a rectal thermostat, heater, and air conditioner are required for each subject), deprives animals of all intentional behaviors and leaves blind tropism without consciousness (as far as I could tell on my visit to Bard's laboratory). Merker cites Bard but he does not cite the work of Goltz, nor of Bures on spreading depression, nor does he cite the distinction between three-layered allocortex and six-layered neocortex, so I pass the question to him: How much of the olfactory and hippocampal cortices remained in the brains comprising his database?

A brain for all seasons

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Abstract: Merker's fine article opens a new view of brain function consistent with current developments in robotics, heuristics, and fuzzy logic. A reciprocal, tripartite organization of input/motivation/output in the midbrain can accomplish the practical tasks of a brain. A bold move places consciousness in the midbrain, raising profound questions about the practical nature of consciousness.

This is a fine article. It opens up a new way to view how brains work. It converges on current developments in robotics, heuristics, and fuzzy logic. As in Brooks' (1986; 1989; 1990; 1991) subsumption architecture, duly noted in this target article, Merker here shows that reciprocal, tripartite organization of input/motivation/output can accomplish the practical tasks of a brain. In this inspiring view, sensory cortex feeds information to midbrain, and midbrain allocates motor resources, and all three act and interact in real time. Merker outlines a reciprocal inside-out/outside-in organization as opposed to the traditional, intractable opposition between top-down and bottom-up. He shows how human neocortex, which is also higher, relative to gravity, can emerge from evolution of more and more powerful sensory, motor, associative, and computational functions, rather than more and more complex executive functions.

In modern times, robots accomplish more and more practical tasks without consciousness. I am among hundreds of thousands of satisfied owners of a relatively inexpensive robot that vacuums floors – backing away from obstacles, following walls, sensing relatively dirty areas for more intensive cleaning, sensing when its battery needs recharging so it needs to stop vacuuming and seek a recharging station. A more advanced model senses proper time to leave its recharging station to start a fresh round of vacuuming. Future models could grease their own bearings or chase away intruders. In a tripartite system such as Merker's, or a subsumption system such as Brooks's, functionality could be added by increasing motor, sensory, and computational capacity in an analogue of the cerebral cortex. The only practical limits would be cost and consumer demand.

Traditionally, both animals in fields and self-interested humans in marketplaces calculate relevant information to arrive at optimal courses of action. In modern times, Gigerenzer et al. (1999, pp. pp. 1–118) and Todd and Gigerenzer (2000) point out that players in field and marketplace rarely, probably never, have access to enough information to arrive at optimal decisions. Moreover, successful action must be prompt action. Prompt action cannot wait to acquire and calculate sufficient information to arrive at an optimum. Gigerenzer and Todd show how players in field and marketplace can take advantage of what they call "fast and frugal" heuristics to arrive at less than optimal, but still useful, decisions.

In field and marketplace, players must divide limited resources among conflicting, often critical, needs. Once again, practical limits of information and time preclude optimal solutions. Meanwhile, fuzzy logic systems, introduced by Zadeh and Kacprzyk (1992), described by Kosko (1993), and applied to industrial and biological examples by Kipersztok and Patterson (1995) and Gardner and Gardner (1998), offer fast and frugal, and

most important, effective and profitable, solutions to problems of apportioning limited resources among conflicting needs.

Erickson (1984) and Erickson et al. (1994) show how modern accounts of color vision can generate the entire visible spectrum with three or only a handful of receptor types, each tuned to a particular wavelength, but each with a band of decreasing sensitivity that overlaps with the others. Erickson (1984) and Erickson et al. (1994) show how this system of relatively few receptors reappears in other modalities. Erickson (1984) also shows how this system of few tuned elements with overlapping bands of sensitivity applies to modern findings of motor systems. Each color, visual angle, taste, and so on, in such systems has a unique code based on the output of a population of receptors. Likewise, each movement in space has a unique code based on a population of afferent outputs. Consequently, efferent and afferent systems can communicate directly and effectively without wasteful intermediary centers. This relieves a midbrain system, such as Merker's, from the burden of centers that must read inputs, translate, and then write outputs, thereby freeing the system to allocate resources among biological needs that realistically fluctuate from moment to moment.

Merker locates consciousness in the midbrain. This is a bold move that raises profound questions about the nature of consciousness. Locating consciousness in a specific structure endows consciousness with a reality that it seldom possesses in cognitive theories. This move faces questions about deciding where, in palpable anatomy, consciousness resides in the brain. This move also faces questions about deciding which beings can exhibit consciousness and which cannot. Brooks and Brazeal (see Brooks 2002, Ch. 8) have raised this question with the robot Kismet with unsettling results. It remains to be seen whether Merker and the parents of infants with cortical birth defects can answer skeptics with firm conviction and subjective observation alone.

Cognitive achievements with a miniature brain: The lesson of jumping spiders

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Abstract: The observation that an animal's behavior is largely unaltered even after profound modifications of sizeable brain portions, suggests a large flexibility in the relationships between species-specific brain structures and species-specific behavior. In this perspective, a fascinating example is given by the comparison of jumping spiders and felids, where similar predatory behaviors are achieved with totally different brain substrates.

The conscious mode of functioning is conceived in the target article as being dependent on specific neural arrangements rather than as being the result of a general increase in informational capacity or complexity achieved by expansion of a structural substrate. This view is in sharp contrast with possible conclusions from studies on self-recognition in mammals. When tested among primates for example, self-recognition – a case of conscious mode of functioning – is observed in great apes and humans, but not in monkeys (Anderson 2001). Among other mammals only large brained cetaceans recognize themselves in a mirror. This capability of self-recognition can be seen as an example of psychological evolutionary convergence with great apes and humans (Delfour & Marten 2001; Reiss & Marino 2001; but see Munger 2006). Moreover, considering that there may be at least a bias for the processing of "self" within the human right prefrontal cortex (Keenan et al. 2000) – a cortical region that, on the basis of examination of the cytoarchitecture, is either absent or very small in cetaceans (Munger 2006) – it could also be argued that self-recognition is a by-product of brain size increase and could indeed be considered as the result of a general increase in informational capacity achieved by expansion of the brain, which below a certain absolute volume does not support self-recognition (see also mirror self-recognition experiments in elephants; Povinelli 1989; Plotnik et al. 2006). This conclusion is somewhat nested in the statement of Merker when he defines reflective awareness as more akin to "a luxury of consciousness on the part of certain big-brained species, and not its defining property" (sect. 1, para. 5). Hence, the definition of consciousness as conceived in the target article is restricted to the state of wakefulness and responsiveness wherein mostly brainstem structures are necessary.

In the framework of this definition, the observation that the behavior of decorticated rats or cats remains from all viewpoints largely the behavior of a rat or of a cat with almost intact cognitive capabilities raises another important issue. Considered from a comparative viewpoint, the various specific behaviors of animals could be understood as adaptative responses of different organisms to dynamic eco-physiological demands. It remains an open research subject to elucidate how specific adaptative behaviors are anchored in specific brains. In other words, is cat brain the only kind of brain that can sustain cat behavior? To what extent does it differ from horse brain, which would be the only brain adequate to sustain horse behavior? The analysis of Merker shows that the competences of decorticate animals reflects the capacity of upper brainstem mechanisms to sustain the behavior required by the adaptations of their species. The fact that this behavior is largely unaltered even after profound alterations of large brain portions suggests a huge flexibility in the relationships between species-specific brain structures and species-specific behavior.

In this perspective, a fascinating example is given by the comparison of jumping spiders and felids. Few terrestrial arthropods catch active prey by stalking them, in the manner of mammalian carnivores. One arthropod group, however, the jumping spiders (*Salticidae*), adopts a strategy in catching prey that is sufficiently similar to that of a cat catching a bird, that is, to creep toward the prey until the chance of escape is small and then spring on the prey. Catching a fly or another spider by stalking is in principle not very different from catching a mouse or a bird. Hence, jumping spiders have evolved a range of visual mechanisms that are remarkably similar to those of predatory higher vertebrates, including complex pattern recognition capabilities. The salticid genus *Portia*, for instance, includes African, Asian, and Australian species that all exhibit complex predatory strategies. *Portia*'s preferred prey is other spiders. The capture of this prey involves behavioral sequences based on performing aggressive-mimicry web signals, problem solving, as well as planning. Flexibility in *Portia*'s predatory strategy clearly characterizes navigation, for which the detouring behavior is particularly illustrative.

Portia routinely reaches prey by taking indirect routes (detours) when direct paths are not available. This even includes detours that require movements initially away from the prey, where the prey is temporarily out of view, or detours and approaches from the rear, when safer, even when direct routes are available (Tarsitano & Andrew 1999). Lions have been observed making such comparable detours when hunting their prey (Schaller 1972). The taking of detours by lions has not been studied experimentally. It can nevertheless be reasonably interpreted as "planning ahead" behavior. The point here is that *Portia*, despite operating with a miniature nervous system, adopts a predatory strategy similar to the one of a lion.

Such an issue is far from trivial. The predatory strategies of *Portia* imply that its visuospatial acuity is more similar to that

of a mammal than to that of an insect, although the size difference is enormous. There are more than 150 million photocells in the human retina, but in a *Portia*'s eye, the photocells number only in the thousands. It is the design of the eight eyes, especially the pair of large forward-facing antero-medial (or "principal") eyes (Figs. 1 and 2), which are responsible for *Portia*'s acute vision (Harland & Jackson 2000). Jumping spiders are not cats however, and their behavioral repertoire for catching prey shows limitations when compared to mammals. A big difference between *Portia* and cats appears to be the speed at which problems are solved. Nevertheless, these limitations only become clearly apparent when the spider is taken out of the natural situation to which it is adapted and made to perform tasks in a laboratory setting. On the other hand, these behavioral limitations are accompanied by an extraordinary degree of neural economy. Arthropods indeed have single cells performing functions that require tens or hundreds in higher vertebrates (Land 1974). More specifically, a salticid spider such as *Portia* makes efficient use of its limited resources for seeing and overcomes many (but not all) of the constraints imposed by its small size. It then is able to achieve considerable cognitive skills, such as problem solving and planning ahead.

In the context of the theoretical implications of the target article, it is of prime interest to know that an animal whose neural machinery is characterized by such a degree of economy also exhibits activities so strikingly similar to those of a mammal.

Figure 1 (Gilissen). *Portia africana*. Size range: 8 to 12 mm. Courtesy Rudy Jocqué.

Figure 2 (Gilissen). *Portia fimbriata*. Size range: 8 to 12 mm. Courtesy Sudhikumar Ambalaparambil.

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I Promethean, bound deeply and fluidly among the brain's associative robotic networks

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Abstract: Merker's insightful broad review fertilely recasts the mind/brain issue, but the phenomenological appeals require additional considerations of behavioral and neural flexibility. Motor equivalences and perceptual constancies may be cortical contributions to a "robotic" tectal orientation mechanism. Intermediate "third layers" of associative neural networks, each with a few diffusely summing convergence-divergence modules, may be the economical expedient by which evolution has extended the limited unity-in-diversity of sensorimotor coordination to perception, action, thinking, and memory.

"*I hope to share with you my fascination with consciousness. Each of you is unique in being at the center of your own awareness, reaching out to the world and other individuals and the stars.*" I begin my biopsychology courses thus inviting awe; then explore the subject of consciousness with student colleagues (Glassman 2002). In 2007 we will read Bjorn Merker's extraordinary synthesis correlating phenomenological consciousness with brain architecture.

Empathy is not enough. Are parts of the article "just-so stories" that conveniently select anatomical or behavioral facts? The first and last sections are fragile in their appeals to empathy, among these, the tormenting ethical "dividing line" issues associated with the touching description of conscious anencephalic children. Are physicians who describe these patients as "vegetative" (sect. 5) attempting a virtuous authoritative role by invoking a mythology to frame pained decisions not to exhaustively engage life-support technology?

We who have even tiny-brained pets like parakeets or goldfish hardly doubt they are conscious. Their behaviors include analogies with anencephalic children's, such as caretaker recognition. Eye-contact empathy occurs especially with anthropomorphic front-eyed pets (Morris 1967, pp. 224–31). Considering Merker's explanation of extreme visual impairment in anencephalic children, his selected Figure 9 photo suggests sham eye-contact based on hearing, as in affectional expressions of children born blind (Eibl-Eibesfeldt 1975, p. 450, Fig. 18.5). Bear in mind that we display related caretaker emotional reactions to dolls and, recently, to high-tech movie animations' uncanny simulations of human facial dynamics, as in Warner Brothers' 2006 film "Happy Feet."

The fact that conscious continuity persisted during Penfield and Jasper's extensive cortical ablations (Penfield & Jasper 1954), says little about localization of consciousness, considering the possibility of rapid compensation or cortical redundancy (e.g., Beach et al. 1960; Glassman & Smith 1988). Analogously, little Parkinsonian deficit may appear until loss of 80% of striatal dopamine terminals (Bezard et al. 2001). Merker's cited instances of absence epilepsy with seizures might be due to loss of tonic arousal rather than a loss of centrencephalic organization.

Visual evolution leads, but "robotic" sensorimotororiented is not enough. Merker argues that mobile visual organization led neurocognitive evolution, with the growing facileness of adaptive recalibrations among topographic sensory mappings. This compelling thesis about the emergence of an *ego center*, around which individuals maintain their own postures in a flux-ridden world, complements the good perspectives of Donald T. Campbell and of Richard Gregory, that evolution of vision became tantamount to *knowing* and *planning*, in freeing organisms to respond to distal stimuli. Vision was the seed for the natural selection of ability to reach deeply into one's past, future, and spatial environment, to "look ahead." Scientists' hypothesis testing, using symbolic thought, evolves naturally from our routine "testing of object hypotheses" in distal perception (Campbell 1956, 1966, 1974; Gregory 1970, 1978).

The spatiotemporal problem of bodily orientation is "intermediate in complexity." More neural machinery is needed to carry it off well than for a segmental reflex, yet enough room for that machinery resides in the narrow hallway of the mesodiencephalon. Yes, there is intriguing *unity-in-diversity* in organisms' ability to orient toward any place within their spheres, but there is also a dull sameness about orienting responses.

Motor equivalence (Milner 1970) and *perceptual constancies* (Rock 1995) comprise more interesting forms of *unity-in-diversity* and more varied, complex relationships between organism and environment – suggestive of consciousness. Such organismic competencies in mediating *patterns* of perception and action have proven most difficult to computerize, like the persistent failure to create a speech machine that emulates ordinary human conversational competence well enough to pass the Turing test (Shieber 2004). Industrial robot arms' graceful orientational movements remain "robotic" in their stereotyped repetitiousness; they achieve organismic flexibility only when telcoperated by a human. Merker may be making a localist error, in placing consciousness in the mesodiencephalic orientation robot, instead of in the larger emergent system.

During the 1960s, watching my advisor, James Sprague (see sect. 3.1) carry out his elegant neurological tests inspired me. In my own later experiments, orientation toward appetitive stimuli sometimes displayed a robotic character, even when visual, auditory, or tactile localizing stimuli could substitute for each other – in cats better than rats (Glassman 1970, 1994). Further evidence that appetitive orientation does not necessarily involve consciousness is in "blindsight" (Weiskrantz 2004). In agreement with some of Merker's points about spherical coordinates (sects. 4.3 and 4.5), an unusual degraded "robotic" orientation response, with dissociated pitch and yaw, appeared during early postoperative days in some cats having large cortical ablations (Glassman 1983). For example, sometimes when a food morsel touched the forepaw of the blindfolded cat, there was first a vertical movement of the snout down to the level of the paw and then a sluggish horizontal turn toward the stimulus side.

Economical connectivity may increase behavioral flexibility. What underlying organization does that "seam" suggest? Sensorimotor behavior normally displays beautiful continuity. An input-output system having few dimensions might save connectivity via data reduction to an intermediate layer of diffusely excitable modules having convergent inputs and divergent outputs. For example, a two-layer network of direct connections between a mosaic of $s = 1000$ distinguishable skin patches and $r = 100$ independently controllable muscle units, requires $sr = 100{,}000$ weighted connections to accurately orient a movement. With an intermediate layer having three summators to integrate input-output associations for three spatial dimensions, $3s + 3r = 3300$ connections suffice (Glassman 1985). The sandwiched associative layer also enhances plasticity, because reciprocal coordinated adjustments in synaptic weights need occur only among the connections of the three modules.

Similar considerations apply to superimposed topographically organized inhibitory circuitry; Merker discusses the economy of such connectivity of the zona incerta (sect. 4.5, Fig. 7). Inhibition can be more diffuse than excitation because damping down responsiveness is inherently less demanding than is achieving accurate threshold, timing, and directionality of an active response. Hence, inhibitory mapping requires less resolution to achieve comprehensive competitive overlap. This yields a safety-factor bonus. Diffuse inhibition makes inaction the default condition, like a "dead man's handle"; foci of excitation have to "break through." A danger in symmetrical "design" of excitatory and inhibitory mappings is that mismatch errors might allow leakage of fragmentary excitation foci, for example, as misplaced sensations, or dyskinesias.

Consciousness in memory extends sensorimotor action organization. Analogous savings considerations might apply to the discussion in section 4.5.2 of cortical long-term memory economy, although most attributes of memory are not literally spatial "dimensions." That is, when a species repeatedly encounters a particular *qualium*, the ability to deftly handle variations of that attribute of its world might evolve more readily if its neural representation were to reify as an independent module, with its own connectivity convergences and divergences. Is *this* what that vast memo-sheet of cortex contains?

In each moment of consciousness immense long-term memory donates a few chunks to working memory (sects. 4.1 and 4.5.2), whose bottlenecked small capacity is robustly similar across species, time scales, and experiential contexts. Small working memory may be a "design factor" limiting combinatorial "explosiveness" (Glassman 1999, 2005); an "ego center" can handle just so much at once. Merker's insight, that the concentration of vertebrate motor outputs caudal to the mesodiencephalon implies that the neural nexus for consciousness is located there, ought to be qualified by noting that we are often quietly thinking. Yet, combinatorial logic must also apply to "cognitive actions." Therefore, evolution of higher cognition may indeed branch from the same slender trunk as has served primitive vertebrates' action-organization.

This wonderfully fertile article has added much to my "to-read" list.

Levels of emotion and levels of consciousness

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Abstract: Merker makes a strong case for the upper brain stem as being the neural home of primary or phenomenal consciousness. Though less emphasized, he makes an equally strong and empirically supported argument for the critical role of the mesodiencephalon in basic emotion processes. His evidence and argument on the functions of brainstem systems in primary consciousness and basic emotion processes present a strong challenge to prevailing assumptions about the primacy of cognition in emotion-cognition-behavior relations.

The central proposition in this commentary is that basic emotions constitute the motivational system ("bias") in the processes of primary consciousness. To relate Merker's conclusion that the mesodiencephalon processes the essential attributes of primary or phenomenal consciousness to compatible emotion theory and research, I will identify two developmental levels or types of emotion and relate them to two levels of consciousness.

Evidence suggests that the mesodiencephalic neural arrangement identified by Merker, through reciprocal connections with other subcortical systems (e.g., amygdala), generate basic

emotion expressions and feelings that play a critical role in organizing primary consciousness and motivating its constituent processes. Basic emotion processes in primary consciousness may help explain the behavior currently attributed to the "new unconscious" (cf. Hassin et al. 2005) and to the "perception-behavior link" (Chartrand et al. 2005).

Emotion schemas. An emotion schema (e.g., love, jealousy, interest in science) represents a dynamic interaction between an emotion and associated perceptions, appraisals, and thought. Emotion schemas emerge in synchrony with cognitive development, and some of them (e.g., shame, guilt) are dependent on a concept of self and on relationships with others (Abe & Izard 1999).

Basic emotions. Basic emotions like joy, sadness, anger, and fear are considered as natural kinds, products of evolution that have a common and universal set of components (neural, bodily/expressive, feeling, action tendency) and characteristics (motivational and regulatory functions) (Izard 2002, 2007 cf. Panksepp 2005b). They can be activated by sensory detection or simple perception of an ecologically valid stimulus and do not require conceptual thought (Ohman 2005). Once activated, they become motivational/functional and regulatory (in terms of target selection and action selection) via rapid, automatic, subcortical information processing, independent of neocortical activity (LeDoux 1996). Thus, they have the characteristics to fulfill the role of the "motivational bias" that Merker identifies in the target selection – action selection sequence mediated by the mesodiencephalic system that supports primary consciousness. The basic emotion of interest is of special significance here. It can be activated by any non-aversive change in the sensory fields, has the capacity to drive and regulate attention and information processing (Lundqvist & Ohman 2005; Silvia 2006), and is critical in the organization of conscious processes and in establishing and maintaining interaction with the social and physical environment (Izard 2007).

Merker's evidence and analysis relating to the brainstem system of primary consciousness indicates that basic positive and negative emotions are well within the purview of children without a cerebral cortex. He identifies expressive behavior patterns in these children that characterize the basic emotions of interest-excitement, joy, and anger in normal infants and young children (cf. Izard et al. 1995).

Levels of consciousness. There is considerable agreement that there is a clear distinction between reflective and primary consciousness (Block 2005; Chalmers 1996; Edelman 2006; Morin 2006; Rosenthal 2002). Reflective consciousness is characterized by symbolic processes, memory, and, ultimately, the capacity for awareness of self and others and for monitoring one's own behavior. As Merker convincingly demonstrates, primary consciousness is characterized by sensory processes that generate subjective feelings (cf. James 1890/1950; Izard 1990), especially emotion feelings, and also includes awareness of and responsiveness to objects in the environment. Apparently, processes in primary consciousness are also critical in early development of normal infants' emotion-expressive/social-communicative behavior that facilitates the forming of social bonds and a network for social support (Shiller et al. 1986; Termine & Izard 1988).

Primary consciousness in normal young infants. The mental processes, particularly the emotion processes, of normal young infants probably operate in primary consciousness, supported by the mesodiencephalon in interaction with the amygdala and hypothalamus. Their cerebral cortex is quite immature and its connections to brainstem systems are still rapidly developing (Bauer 2006; Greenough 1991; Posner & Rothbart 2000). Nevertheless, 3-day-old infants can discriminate their mother's voice and work to produce it (DeCasper & Fifer 1980). Three- to 4-month-old infants can form concepts, (Quinn et al. 2001), and 6-month-old infants can form associations between memory representations that are absent (Cuevas et al. 2006). Of course, young infants (0–9 months) are incapable of long-term memory, higher-order cognition, and self-awareness (Bauer 2006; Lewis et al. 1983), and hence cannot engage in the processes of reflective consciousness.

Emotion processes in primary consciousness. From a developmental perspective, it is expectable that emotion expressions and behavioral activities of normal young infants would be similar to those of children without a cerebral cortex. The effects of the emotion expressive behavior of these contrasting groups of children have similar effects on parents and enhance the development of meaningful parent-child relationships. A child without a cortex cannot regulate emotions efficiently or exercise cognitive control of emotion-expression or emotion-related behavior. The same is true of normal young infants. They depend almost entirely on non-cognitive processes for soothing or regulation of intense/run-away emotions following the acute pain of inoculation (Izard et al. 1987).

Emotion prowess in primary consciousness. Four-month-old infants can discriminate and respond differentially to discrete positive and negative emotion expressions of their mothers (Montague & Walker-Andrews 2001), an ability that will eventually facilitate empathic responding. Even 3-month-old infants often take the initiative in displaying and responding with emotion when their mother makes a poker face and remains still and silent (Hembree 1986; Tronick & Cohn 1989). Such expressive-behavior play is fundamental to the development of emotion knowledge (the understanding of the expressions, feelings, and functions of emotions) that will eventually become critical to the development of interpersonal skills and the prevention of psychopathology (Denham & Burton 2003; Izard 2002).

Emotion processes in primary consciousness in adults. Evidence suggests that a brainstem-amygdala network mediates the activation and expression of basic emotions in human adults (Ohman 2005). The behaviors facilitated by brainstem mechanisms in primary consciousness may bear some similarity to behavior currently attributed to "nonconscious" or "unconscious" cognitive and emotional processes in normal adults.

It is speculative to compare psychology's "new unconscious" (Hassin et al. 2005) and "perception-action link" (Chartrand et al. 2005) with processes in primary consciousness (Block 2005; Edelman 2006). Nevertheless, they clearly have a central feature in common: they both involve unreportable mental processes (including emotion processes) that affect behavior. Processes mediated by brainstem or brainstem-amygdala circuits generate "unconscious" emotion feelings that affect behavior in observable ways (Ohman 2005; Winkielman & Berridge 2004). Attributing causal roles to emotion processes in primary consciousness may be more straightforward and more heuristic than attributing causal roles to the "unconscious" and particularly to "unconscious emotions."

Concluding remarks. The term primary or phenomenal consciousness as defined by Merker and others may provide a better descriptor for some of the processes currently attributed to the "unconscious," and particularly to "unconscious emotion" (cf. Panksepp 1998a). Attributing emotion feeling to primary consciousness means that one can become conscious of a feeling that one cannot label and articulate (cf. Bruner et al. 1956), as demonstrated in normal infants and children without a cerebral cortex, and hypothesized to be the case for anyone (Izard 1991).

The tendency in psychology has been to assume that mental processes operate either in reflective consciousness or in an "unconscious domain," neither of which explicitly correspond to or adequately frame the processes of primary consciousness described by Merker and a number of philosophers and scientist-philosophers (e.g., Block 2005; Edelman 2006; Rosenthal

2002). Lack of a clear differentiation among processes in primary consciousness and in other levels of mental functioning may add to confusion and slow the development of scientific interest in the subject. Merker's target article presents a strong challenge to the prevailing notion of cognitive primacy in emotion processes and in emotion-cognition-behavior-relations (cf. Zajonc 1980).

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Target selection, attention, and the superior colliculus

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Abstract: Consistent with the target article, recent evidence indicates that the superior colliculus (SC) is somehow involved in target selection. However, it is not yet known whether this function is inherent to the SC or inherited from its inputs, how the selection process occurs for different movements, or how target selection by the SC is related to covert selection (i.e., attention).

It has been recognized for some time that the intermediate and deep layers of the superior colliculus (SC) in primates plays some role in target selection, at least for saccadic eye movements. For example, the preparation of saccades is correlated with increases in the activity of SC neurons that can begin hundreds of milliseconds before any movement and this activity appears to play a role in representing possible targets (Glimcher & Sparks 1992). Changing the probability that a visual stimulus will be the target – for example, by adding a variable number of irrelevant stimuli – changes the visual and tonic activity of many SC neurons (Basso & Wurtz 1997; Dorris & Munoz 1998). When the subject must search for a uniquely colored target stimulus amidst other colored distracters, many SC neurons discriminate the target from the distracter with a delay that is time-locked to stimulus onset, rather than saccade onset, suggesting that they play a role in target selection in addition to saccade preparation (McPeek & Keller 2002).

Perhaps the most compelling evidence for a role of the SC in target selection, as distinguished from saccade selection, comes from studies of the other type of voluntary eye movement made by primates – smooth pursuit. The SC has long been known to contain a motor map for saccades, but more recent studies have shown that the activity of many saccade-related SC neurons is also modulated during pursuit eye movements. These neurons show a somewhat complicated temporal pattern of activity during pursuit – and also fixation – but this pattern can be explained fairly simply by considering the location of the tracked target within the neurons' retinotopically organized response fields (Krauzlis et al. 1997, 2000). The distribution of activity across the SC motor map therefore appears to provide a real-time estimate of the position of the target in oculocentric or retinotopic coordinates, not restricted to saccades but for orienting movements in general. This "target position map" hypothesis provides what we consider to be a parsimonious alternative to the widely discussed "fixation zone/saccade zone" hypothesis (Munoz & Fecteau 2002), but the issue remains controversial.

The activity of many SC neurons also predicts the subject's choice of target for pursuit as well as for saccades. During a visual search task, many SC neurons exhibit a preference for the target stimulus over irrelevant distracters that emerges over the course of ~100 ms prior to the initiation of pursuit and saccades (Krauzlis & Dill 2002). By interpreting the preference for the target stimulus as a "decision signal," we showed that SC activity could account for the target choices made by pursuit and saccades. We also inferred that pursuit uses a less stringent decision criterion than saccades, perhaps because errant saccades are more costly in their disruption to vision than mistakes by pursuit. These physiology results have been recently corroborated by behavioral studies in human subjects showing evidence that pursuit and saccade choices are guided by a common decision signal, and that the decision to trigger pursuit involves a threshold that is generally lower that that for saccades (Liston & Krauzlis 2003; 2005). The idea of a common decision signal is consistent with the integrative viewpoint put forward in the target article, but these issues are also not yet settled. For example, an alternative viewpoint is that target selection involves a serial linkage between saccades and pursuit, with pursuit simply adopting the choice made by the saccade system (Gardner & Lisberger 2002).

A pair of studies has recently demonstrated the idea that the SC is causally involved in target selection. The first study, focusing on saccadic eye movements, used a visual search task and found that when the region of the SC representing the target was focally inactivated, saccades were often misdirected to distracters appearing in unaffected areas of the visual field (McPeek & Keller 2004). The second study examined both saccades and pursuit using a luminance discrimination task and found that subthreshold microstimulation of the SC biased the selection of targets toward the stimulated location for both types of eye movements (Carello & Krauzlis 2004). The results for pursuit were especially revealing. Because the targets for pursuit initially appeared at a location opposite to its direction of motion, the experiment was able to distinguish between effects on the motor commands (i.e., which direction to move) and effects on the position of the target (i.e., which stimulus to follow). The results showed that altering SC activity changed which stimulus was chosen, regardless of the type or direction of eye movement that was needed to acquire the target. These experimental results provide strong support for the interpretation put forward in the target article that the primate SC plays an integrative role in target selection and decision-making, beyond its conventional role in the motor control of saccades.

What remains unclear from these studies is the extent to which target selection is a function that is inherent to the superior colliculus, a point that is central to the "mesodiencephalic" theory of consciousness put forward in the target article. A fairly common view of these recent findings is that the SC functions as a conduit for selection signals that are generated in other places, such as the cerebral cortex. Unfortunately for the theory, it is difficult to rule out this interpretation, because the extensive cortical and subcortical network involved in target selection makes it difficult to isolate the contribution of individual brain regions. Nonetheless, one crucial test is to determine how the inactivation of various cortical areas involved in target selection alters the properties of neurons elsewhere in the network, including the SC. These experiments would most likely identify multiple sources of support for target selection, but they might also help identify how the basic form of target selection putatively accomplished by the SC is extended in functional scope by the addition of signals from the forebrain.

A better test of the theory is suggested by the strongest prediction put forward in the article, namely that "*one conscious content will not be replaced by another without involvement of the mesodiencephalic system (centered on the superior colliculus) as outlined here, even when that change is unaccompanied by eye movements*" (sect. 4.5.1, para. 4, emphasis in original). Testing the contents of consciousness in animal subjects poses serious

challenges, but some recent studies have shown that stimulation in the SC alters performance in ways that mimic visual attention (Cavanaugh & Wurtz 2004; Muller et al. 2005). It is premature to conclude from this evidence that the SC plays a causal role in determining the contents of perceptual awareness, first because "attention" is not synonymous with "awareness," but also because the effects of the stimulation likely extend to a network of areas connected to the SC, including several cortical areas that are themselves implicated in the control of attention. However, similar tests of visual attention can be conducted using selective inactivation of SC neurons, as has been done for target selection. Such experiments would provide an important test of the "mesodiencephalic" theory, and indicate whether further tests seem worthwhile.

In summary, the target article presents a provocative and contrarian theory of consciousness, but one that is supported by recent experimental findings about the role of the primates SC in target selection. Even more importantly, the theory makes specific predictions about the role of the SC in the control of perceptual awareness that could be tested experimentally.

Consciousness is more than wakefulness

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Abstract: Merker's definition of consciousness excludes self-reflective thought, making his proposal for decorticate consciousness not particularly ground-breaking. He suggests that brainstem sites are neglected in current theories of consciousness. This is so because broader definitions of consciousness are used. Split-brain data show that the cortex is important for full-blown consciousness; also, behaviors exhibited by hydranencephaly patients and decorticated rats do not seem to require reflective consciousness.

In the target article Merker wisely starts by explaining what his view of consciousness is. He defines consciousness as "a state of wakefulness . . . which typically involves seeing, hearing, feeling, or other kinds of experience" (sect. 1, para. 1) but excludes reflective awareness (i.e., being "aware that one is seeing, hearing, and so forth"; sect. 1, para. 6). As such, consciousness is equated with wakefulness and responsiveness to one's environment, and the reader is indeed tempted to concur with the author that consciousness results from activity of subcortical and brainstem mechanisms. In other words, the proposal that consciousness, as defined here, is possible without a cortex does not seem particularly ground-breaking and has been supported by neurophysiological evidence for quite some time now (as Merker extensively documents in the target article).

Merker states that "Few cognitivists or neuroscientists would today object to the assertion that 'cortex is the organ of consciousness'" (sect. 1, para. 7). "With some notable exceptions [. . .], brainstem mechanisms have not figured prominently in the upsurge of interest in the nature and organization of consciousness that was ushered in with cognitivism in psychology and neuroscience" (sect. 1, para. 7). This is not surprising, since what most researchers today are interested in is not "consciousness in its most basic and general sense, that is, as the state or condition presupposed by any experience whatsoever" (sect. 1, para. 2), but in full-blown introspective consciousness – which *does* depend on cortical activity. More than forty-five years of split-brain research has convincingly shown that surgically isolating the cerebral hemispheres alters consciousness (Gazzaniga 2005). At least six main interpretations of commisurrotomy have been put forward (Morin 2001) – of which only one suggests that consciousness is unaltered by the surgical procedure; the other five views (pre- and post-operation dual consciousness, equal and unequal division of consciousness, and dual personhood in the intact brain) all ascribe a key role to the cerebral hemispheres (and thus to the cortex) in consciousness. The fact that Merker does not mention this large body of work in the target article is rather disconcerting.

Hydranencephaly is used by the author to support his view of decorticate consciousness. He reports his first-hand experience with children afflicted by this condition and proposes that "These children are not only awake and often alert, but show responsiveness to their surroundings in the form of emotional or orienting reactions to environmental events" (sect. 5, para. 6). This is followed by a description of behaviors that these children can engage in, including expressing pleasure and aversion, differentially responding to the voice of familiars, showing preferences for situations, and taking behavioral initiatives. It is further observed that decorticated rats can "stand, rear, climb, hang from bars, and sleep with normal postures" (sect. 4.4, para. 2). They can also swim, eat, mate, and defend themselves. The question, of course, is: How should one interpret such behaviors in relation to consciousness? Does expressing emotions or swimming entail "consciousness" as defined by Merker? Certainly. Do these behaviors necessitate self-awareness? Most probably not. This represents a challenge reminiscent of the one primatologists face when trying to determine if apes possess Theory-of-Mind, autonoetic, or metacognitive abilities (see Terrace & Metcalfe 2005). For instance, one can ask animals to recall food locations or past personal events to test autonoetic consciousness. Monkeys can indeed exhibit such behaviors (Menzel 2005; Schwartz 2005), but again, the point is that such behaviors most likely imply wakefulness and responsiveness, but not reflective consciousness.

Merker cites Baars (1988), Mandler (1975), and Miller (1986) as examples of theorists who do not focus on subcortical brain areas in their attempts to explain consciousness. The reason for this is simple: their definition of consciousness is much broader than the one proposed in the target article. To illustrate, Baars' definition of consciousness (1988) includes one's private experience of reading a word, remembering what one had for breakfast yesterday, and the feeling of a toothache – that is, instances of visual and auditory images, inner speech, bodily feelings, and so forth. Consciousness also contains "peripheral" information at the fringe of conscious experience – for example, the vague awareness one has of surrounding noises. Consciousness also encompasses one's access to current beliefs, intentions, meanings, knowledge, and expectations, as well as voluntary control. Baars' more operational definition of consciousness requires that (1) the organism can testify that it was conscious of something following the conscious experience, and (2) an independent effort at verifying the accuracy of the experience reported by the organism be made. Interestingly, Baars rightly notes that in reporting its experience the organism engages in a metacognitive act. Clearly, such a view of consciousness goes far beyond wakefulness and incorporates autonoetic consciousness (access to one's autobiography and mental time travel), self-description, verbal report, metacognition, and self-agency. These various facets of consciousness are reflective in essence.

If one defines consciousness simply as a state of wakefulness and responsiveness, then of course only brainstem sites are necessary, and Merker's careful analysis is very useful in that respect. However, if one embraces the more common view of consciousness which includes self-reflection (e.g., Dennett 1991; Schooler 2002; Zelazo 1999), then obviously cortical areas are involved (e.g., Craik et al. 1999; Goldberg et al. 2006; Johnson et al. 2002; Kjaer et al. 2002), and Merker's thesis does not apply.

Supracortical consciousness: Insights from temporal dynamics, processing-content, and olfaction

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Abstract: To further illuminate the nature of conscious states, it may be progressive to integrate Merker's important contribution with what is known regarding (a) the temporal relation between conscious states and activation of the mesodiencephalic system; (b) the nature of the information (e.g., perceptual vs. premotor) involved in conscious integration; and (c) the neural correlates of olfactory consciousness.

Evidence from diverse sources has led to the consensus that conscious states integrate neural activities and information-processing structures that would otherwise be independent (Baars 2002; see review by Morsella 2005), but no such agreement has been reached regarding which neuroanatomical regions underlie this special form of integration. By reexamining long-overlooked neurological findings, Merker elegantly isolates subcortical regions that may give rise to these elusive states. With this important contribution in mind, it may be progressive to evaluate whether the temporal dynamics of these subcortical (albeit "supracortical") events are consistent with what has been documented regarding the substantial delay between afference from exteroceptors and its consciously experienced effects (see review by Libet 2005). Does activation from a supraliminal stimulus influence the mesodiencephalic system at the same time that an associated change in consciousness is predicted to occur (e.g., several hundred milliseconds following stimulus presentation; Libet 1986)? Given how much is known regarding the processing speed of the hardware at hand (e.g., neurons and synapses) and about the timings of different *stages-of-processing* as gleaned from psychophysiological recordings, answering this question may be a feasible way to obtain additional corroboratory evidence for Merker's framework. Moreover, such evidence may be in agreement with the claim that the contents of conscious states reflect the final product of a relatively timely process in which multiple, consciously impenetrable interpretations or "drafts" of sensory afference and other forms of information are entertained and evaluated (Dennett 1991).

In addition, it may be informative to evaluate whether the nature (e.g., perceptual, semantic, premotor, or motor) of the kinds of processes occurring in these integrative, supracortical regions is consistent with the view that conscious states are necessary to integrate only certain kinds of information. It is clear that many kinds of information can be integrated without these states. For example, consciously impenetrable interactions are exemplified in countless intersensory phenomena, including McGurk (McGurk & MacDonald 1976) and ventriloquism effects (Vroomen & de Gelder 2003). Indeed, it has been recently proposed that neocortical operations are essentially multisensory in nature (Ghazanfar & Schroeder 2006). That such neocortical interactions can be unconscious is consistent with Merker's proposal that cortical processes are not the seat of conscious states.

In line with Merker's "premotor" characterization of these supracortical processes and with his characterization of the "final common path," Supramodular Interaction Theory (SIT; Morsella 2005) proposes that conscious states are necessary to integrate specific, multimodal systems that are unique in that they may conflict with skeletal muscle plans, as described by the *principle of parallel responses into skeletal muscle* (PRISM). In harmony with Merker's account, these systems are defined by their concerns (e.g., bodily needs) and skeletomotor goals rather than by their sensory afference, the latter being the traditional way in which mental faculties have been characterized (Ghazanfar & Schroeder 2006). SIT illuminates why conscious states are required to integrate some processes (e.g., "pain-for-gain" scenarios as when carrying a hot plate of food or holding one's breath) but not others (e.g., intersensory interactions, peristalsis, and the pupillary reflex), and explains why skeletal muscles have been regarded as "voluntary muscles." Skeletal muscles are at times "consciously controlled" because they are directed by multiple systems that require conscious states in order to interact and collectively influence action. Accordingly, regarding processes such as digestion, one is conscious of only those phases of the processes that require coordination with skeletal muscle plans (e.g., chewing or micturating) and none of those that do not (e.g., peristalsis). Together, these proposals are consistent with the view that the properties of conscious states are intimately related to action production (Barsalou 2003; Glenberg 1997; Hommel et al. 2001; Sperry 1952), a view that challenges traditional accounts that divorce input from output processes (cf. Eimer et al. 1995).

Also consistent with Merker's account is the extensive research on "split-brain" patients and on binocular rivalry (cf. O'Shea & Corballis 2005), which strongly suggest that the minimal anatomy for a conscious brain does not require the cerebral hemispheres, nor the commissures (or transmission processes) connecting them. Moreover, although extirpation of the amygdalae and hippocampi lead to anomalies including severe deficits in affective memory (LeDoux 1996) and episodic memory (Milner 1966), respectively, it seems that an identifiable and reportable form of consciousness persists without either of these structures. It seems as well that such a minimal, conscious brain does not require interactions between the afferent impulses from the sensory organs and the initial "relay" at the thalamus, for one experiences aspects of olfaction consciously even though the signals from the olfactory sensory system bypass the thalamus and directly target regions of the ipsilateral cortex (Shepherd & Greer 1998). Of course, this does not imply that a conscious brain experiencing only olfaction does not require a thalamus. Consistent with Merker's account, in subsequent, postcortical stages of processing, the thalamus does receive inputs from cortical regions that are involved in olfactory processing (Haberly 1998).

Hence, because of its neuroanatomical accessibility and its relatively simplistic and phylogenetically primitive arrangement (Shepherd & Greer 1998), the olfactory system may prove to be a fruitful system in which to further isolate the neural processes giving rise to conscious states within the mesodiencephalic regions already identified by Merker. According to Buck (2000, p. 633), conscious aspects of odor discrimination depend primarily upon the activities of the frontal and orbitofrontal cortices, a proposal which, at least at first glance, seems inconsistent with Merker's primarily "subcortical" account of the neural correlates of conscious states. Additional research on olfactory consciousness and the olfactory components of mesodiencephalic regions may reconcile both views and thus further our understanding regarding the general nature of the physical substrates of conscious states.

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Subcortical regions and the self

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Abstract: Merker argues that subcortical regions are sufficient for the constitution of consciousness as "immediate, unreflective experience" as distinguished from self-consciousness. My point here is that Merker neglects the differentiation between pre-reflective self-awareness and reflective self-consciousness. Pre-reflective self-awareness allows us to immediately and unreflectively experience our self, which functionally may be mediated by what I call self-related processing in subcortical regions.

Merker argues in his article that subcortical regions are sufficient to constitute consciousness, which he defines as the ability to experience. He calls this ability "immediate, unreflective experience" (sect. 1, para. 5). This must be distinguished from states where one is aware that one is experiencing something, which he calls "additional awareness," "reflective consciousness," "reflective awareness," or "self-consciousness." He considers the cortex to be necessary only for reflective consciousness but not for consciousness as "immediate, unreflective experience." The focus is thus on subcortical regions like the substantia nigra (SN), ventral tegmental area (VTA), superior colliculi (SC), raphe nuclei (RN), hypothalamus (Hy), midbrain reticular formation (MRF), and the periaqueductal grey (PAG). The aim of my comment is to complement Wickers' notion of consciousness as being "immediate, unreflective experience" by what I, in orientation on phenomenology (Husserl 1991; Zahavi 2005), call "pre-reflective self-awareness."

Recent imaging studies have focused on the self and observed predominantly cortical midline regions to be associated with high degrees of self-relatedness (see Northoff & Bermpohl 2004; Northoff et al. 2006). Though surprisingly little has been reported of subcortical regions (probably in part because of methodological reasons), some studies have observed their involvement in self-relatedness. Phan et al. (2004), for example, observed the association of the ventral striatum/N.accumbens (VS/NACC) with self-relatedness of emotional pictures. Similar observations with additional recruitment of the tectum, the PAG, the dorsal medial thalamus, and the colliculi have been made by Schneider et al. (submitted), though these researchers, unlike others, did not include an explicit cognitive component (e.g., decision about self-relatedness) in the activation paradigm itself. The involvement of these subcortical regions – especially the PAG and the tectum, including the SC – in processing self-relatedness has also been postulated by Panksepp (1998a; 1998b; 2003; 2005a). Based on their connectivity pattern in receiving both multiple sensory and motor afferences/efferences, these regions may be crucial in "relating" sensory and motor stimuli to the organism itself. The process of "relating" presupposes what I call self-related processing (Northoff & Bermpohl 2004; Northoff et al. 2006). Self-related processing concerns stimuli that are "experienced" as "strongly related" to the organism in its respective environmental context. "Experience" refers to the subjective aspect of experience, which is described as the "phenomenal aspect" (Block 1996; Chalmers 1996) and must be considered prereflective as distinguished from reflection, for example, cognitive aspects – this mirrors what Merker calls "immediate, unreflective experience." The term "strongly related" points out the process of associating and linking intero- and exteroceptive stimuli with a particular organism or person. The more the respective stimulus is associated with the person's sense of belongingness, the more strongly it can be related to the self. Ultimately, the self-stimulus relation results in the "immediate, unreflective experience" of what has been called "mineness" or an "addition of the 'for me'" (Lambie & Marcel 2002). What I immediately and unreflectively experience is therefore not only the stimulus itself, consciousness, but also, at the same time, myself as it is related to the stimulus – this has been called pre-reflective self-awareness. Accordingly, if subcortical regions are supposed to mediate consciousness, they may also mediate the co-occurring pre-reflective self-awareness that may explain the aforementioned involvement of these regions in imaging studies of self-relatedness.

What exactly happens in self-related processing? How can we characterize the term "process"? Instead of comparing stimuli with an absolute measure of self-relatedness, as reflected in a fixed and predefined self, stimuli are compared and matched with each other in terms of their fit and accordance. Certain interoceptive stimuli fit and match well with particular exteroceptive stimuli, whereas they do not fit well with others. For example, a highly aroused stress system causing a person excitement does not match with a rather calm and relaxing environment – the person will consequently have some difficulties in relating to this environment, which will therefore be designated as rather poorly self-related. If, in contrast, the person wants to relax and calm down, such an environment will be designated as highly self-related. Accordingly, self-related processing describes the matching and comparison between intero- and exteroceptive stimuli. This corresponds nicely to Merkers' description of the interaction between action (body), target (world), and needs (motivation), which he links with subcortical regions and which are, according to him, matched with each other. He assumes a sensorimotor-based ego-center to be the result of this matching process and distinguishes it from what he calls self-consciousness. Here I want to differentiate his terminology. What Merker describes as a sensorimotor based "ego-center" corresponds to what I and phenomenologists call pre-reflective self-awareness, an immediate and unreflective experience of the bodily based organism or person within the world. Whereas what Merker describes as self-consciousness may be more correctly termed "reflective self-consciousness."

Finally, Merker illustrates his hypothesis with the example of people without cortex, so-called hydrancephaly. His impressive description of these patients illustrates another aspect of self-relatedness, as characterized in a pre-reflective way. These patients are well able to react to salient stimuli in their environment, especially to those they are particularly familiar with, such as their parents. Self-relatedness may thus be considered a special instance of salience in general, for example, social salience. By matching intero- and exteroceptive stimuli with regard to their belongingness to the person, self-related processing allows the person to react to and navigate within a given environment and distinguish its various components according to their social salience. The case of hydranecephalic patients thus illustrates that self-related processing may be considered nothing but social salience, and that it can be well preserved even if one is not aware of it as such. Accordingly, self-relatedness, allowing for social salience and navigation within the environment, must be considered more basic and fundamental than the awareness of one's ability to experience oneself and to navigate within one's environment. This means in neural terms that subcortical regions are essential for consciousness and pre-reflective self-awareness because otherwise no self-related processing would be possible. Although cortical regions allowing for our ability to become aware of consciousness and pre-reflective self-awareness may be considered an additional function that allows me to write this comment about the self, this, however, is not absolutely necessary for my ability to constitute self-relatedness as social salience.

Emotional feelings originate below the neocortex: Toward a neurobiology of the soul

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Abstract: Disregard of primary-process consciousness is endemic in mind science. Most neuroscientists subscribe to ruthless reductionism whereby mental qualities are discarded in preference for neuronal functions. Such ideas often lead to envisioning other animals, and all too often other humans, as unfeeling zombies. Merker correctly highlights how the roots of consciousness exist in ancient neural territories we share, remarkably homologously, with all the other vertebrates.

A cortical view of consciousness has become so prevalent that several generations of research related to the subcortical foundations of consciousness almost disappeared from reasoned discourse during the last few decades. Merker provides a long-overdue corrective. He envisions how brainstem functions are foundational for phenomenal experience as being more than simply arousal.

Consciousness is not critically related to being smart; it is not just clever information-processing. Consciousness is the experience of body and world, without necessarily understanding what one is experiencing. Primary phenomenal states have two distinct but highly interactive branches: (1) the ability to perceive and orient in the world, and (2) the ability to feel the biological values of existence. Merker has focused on the former. I will focus on the latter – the primary *affects*, from bodily hungers to emotional delights. If we get the foundational issues right, then the secondary and tertiary layers of consciousness – the ability to have thoughts about the world and thoughts about thoughts – should become easier hard problems.

Merker highlights subcortical regions as affecting primary process perceptual consciousness. Let me try to illuminate raw affective experience. Is either of these more fundamental? In mind evolution, were the perceptual or the motivational-emotional components more essential for the emergence of experiential capacities within brains? I would choose core motivational and emotional brain processes that symbolize bodily values – the diverse rewards and punishments that guide behavioral choices allowing organisms to seek comfort zones that promote survival and avoid discomfort zones that hinder survival. I suspect the more ancient, medially concentrated interoceptive motivational-emotional urges of the brainstem were foundational for the more lateral zones that harvest external information for guidance of behavior. Primary consciousness in Shewmon et al.'s (1999) neurologically impaired children was most dramatically evident in their affective presence.

Within the meso-diencephalic continuum, damage to the medial components, such as the periaqueductal gray and surrounding reticular zones, impairs consciousness more than comparable damage to surrounding tissues that process exteroceptive inputs (Panksepp 1998a; 1998b). This makes evolutionary sense if consciousness was premised on fundamental survival issues, related quite directly to organismic integrity. The most vital (least expendable) parts of the body are the viscera, neurosymbolically concentrated in centromedial regions of the mesencephalon and in the hypothalamus. Thus, neuroscientists since Hess (1957) and MacLean (1990) onward have accepted the existence of a visceral nervous system, which detects and behaviorally elaborates bodily needs. Very medial homeostatic detectors (i.e., for hunger, thirst, etc.) regulate adjacent core emotional systems that generate many distinct instinctual-emotional "intentions in action" – to use Searle's (1983) felicitous phrase.

Had William James known about such ancient brain layers, he might never have envisioned emotional feelings emanating from peripheral autonomic commotions perturbing cognitive regions of the brain – a theory that has captivated psychology to the present. Even Damasio's (1994) related somatic-marker hypothesis – placing *emotional* feeling within somatosensory cortex – remains a weak working hypothesis. That many *sensory* feelings are elaborated in insula is now well accepted. So far, there is little evidence that *peripheral bodily indices* of emotions *precede* and *cause* emotional feelings which control decision-making (Hinson et al. 2006). Had James known of the visceral-limbic brain, surely he would have considered that those networks generate emotional feelings directly (Panksepp 1998a; 2005a). Likewise, as far as we know, no Jamesian "mind-dust" permeates the universe.

If we envision three key mesencephalic-diencephalic functions as concentric circles, with (1) body need detectors situated most medially (Denton 2006), (2) emotional-instinctual systems concentrated in subsequent layers (Panksepp 1998a), and with all surrounded by (3) more externally directed somatosensory and somatomotor processes for attentive target selection and directed actions, we have a working image of primary process phenomenal consciousness. Affective consciousness, comprised heavily of the two highly interactive medial layers, may suffice for some level of experience, probably without self-awareness. Surely those emotional-instinctual layers are of foremost importance for psychiatry (Panksepp 2006). With brain maturation, additional cognitive complexities emerge upon the solid foundation of the more ancient primary processes, as Merker highlights with the compelling Sprague effect. Clearly, raw consciousness survives damage to neocortical sensory and motor homunculi.

All mind scientists should remember: Primary consciousness arises from the somato-visceral operating systems of the upper brainstem (Watt & Pincus 2004). There is something deeply personal about this kind of neural activity. This is where our bodily needs are felt (Denton 2006). These brain-mind abilities imbue experience with ownership. Perhaps subtle body representations permeate these networks of primary-process consciousness. Sensory homunculi have understandably lost appeal as necessary substrates of experience, and not just because of the illogic of infinite regress of observers. But more diffuse visceral sensory-motor integrative homunculi exist in lower regions of the brain. Just as Merker needs an "ego-center" at the core of phenomenal experience, I need a coherent core-SELF (Simple Ego-type Life Form) – a neurobiological action "soul" – as a foundation for experienced existence (Panksepp 1998a, 1998b).

There is currently little neuroscientific work on the biology of the soul, but a special issue of the *Journal of Comparative Neurology* (2005, 493:1–176), intriguingly entitled *The Anatomy of the Soul*, focused well on the subcortical depths of bodily functions, spiced with some discussion of mentality. Why do most neuroscientists remain impaled on the dilemma of how mental experience could ever emerge from physiochemical processes of the brain? This dilemma has engendered a most ruthless reductionism – where neuro-mental properties are rarely evident in discussions of what other animals do. Thus, neo-neurobehaviorism still rules: In mainstream neuroscience, other animals are generally regarded as little more than zombies. But this is an ontological presumption rather than an epistemological likelihood. By the weight of empirical evidence, all other mammals are sentient beings (Panksepp 2005a). And if we do not learn how to investigate these issues in animals, we will never have a detailed science of consciousness. It is noteworthy that the centrencephalon vision emerged first from animal research. Hopefully, Merker's powerful thesis will restore such eminent concepts to consciousness studies.

Since we still live in ruthlessly reductionistic times, let me close with a few anecdotes. When we discovered an abundant ultrasonic vocalization (USV) during rat play, we eventually conceptualized this social joy response as an ancestral form of laughter (Panksepp & Burgdorf 2003). When we first sought publication in *Nature*, a famous fear-conditioning researcher torpedoed us with this remark: "Even if their interpretation were true, they will never be able to convince their colleagues." We eventually published the ruthlessly rejected work elsewhere (Panksepp & Burgdorf 1999).

After we discovered that even complex behaviors such as play survive radical neo-decortication (Panksepp et al. 1994), I stumbled on something quite special in an undergraduate neuroscience lab. Sixteen students were each given two *adult* animals, one of which was neurologically intact, the other neo-decorticated at three days of age. After two hours of free

observation, the students had to decide which was which. Twelve of the decorticates were identified as normals – a statistically significant mistake! Why? Because decortication had released primary process emotionality! ... a phenomenon known since the late 19th century. Decorticates are more active, more apparently engaged, sometimes enraged, with the world. Our neurologically intact rats were more inhibited and timid (worries on their mind?).

I trust that Merker's astute analysis will not fall on deaf ears among many investigators who believe that *awareness* (knowing you experience) is the *sine qua non* of consciousness. We can all agree on the facts. When practically all higher-brain regions are removed in animals (Kolb & Tees 2000; Panksepp et al. 1994) or congenitally absent in human children (Shewmon et al. 1999), core consciousness survives. Such organisms exhibit a remarkable emotional vitality of behavior, and it is our responsibility to entertain that mentality still exists in the remnants of their brains. A science that burrows its head opportunistically in the sand is a second-rate science.

If we wish to scientifically understand the nature of primary-process consciousness, we must study the subcortical terrain where incredibly robust emotional and perceptual homologies exist in all mammalian species. Without work on animal models of consciousness, little progress, aside from the harvesting of *correlates*, can be made on this topic of ultimate concern. I appreciate Merker's timely reminder about the history of our discipline, and the need for a better understanding of animate life on earth, than any form ruthless reductionism provides.

The ontology of creature consciousness: A challenge for philosophy

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Abstract: I appeal to Merker's theory to motivate a hypothesis about the ontology of consciousness: Creature consciousness is (at least partially) constitutive of phenomenal consciousness. Rather than elaborating theories of phenomenal consciousness couched solely in terms of state consciousness, as philosophers are fond of doing, a correct approach to phenomenal consciousness should begin with an account of creature consciousness.

A traditional question about consciousness is whether preverbal children have phenomenal experiences, and if they do, what convinces us that they do. In this context, congenitally decorticate children are not even considered worth discussing. Yet Merker argues that (some) children with hydranencephaly have phenomenal experiences. He backs up his claim with an elaborate theory supported by a wide range of evidence. To make sense of his theory, we might need to think about the ontology of consciousness in a new way.

When philosophers attempt to spell out what consciousness is, they typically formulate the problem in terms of so-called *state consciousness*: What does it take for a mental state to be an experience? Their most worked-out answers employ two kinds of ingredients: functional and representational. Their least worked-out answers appeal to some condition to be discovered empirically by scientists. For instance, pain might be C-fiber firing, or whatever scientists tell us. Well, Merker is a scientist, and he is telling us something.

Merker tells us that "primary consciousness" has the function of integrating sensory information and motivations to select targets and actions. He adds that primary consciousness is constituted by the structure of the "analog reality simulator" that fulfills this function. This may sound like a hybrid functional-representational theory. But Merker's theory does not say what it takes *for a mental state* to be conscious. It is not even formulated in terms of mental states. Furthermore, Merker attributes consciousness to some congenitally decerebrate children. How plausible is it that such children have experiences as we do? If we keep framing the question of consciousness in traditional terms – that is, in terms of what it takes for mental states to be phenomenally conscious – we seem to face a dilemma: Either decorticate children have the same kind of conscious states that we have, and hence have phenomenal consciousness, or they don't, and hence have no phenomenal consciousness. Either way, Merker has not told us what it takes to have such states. We can dismiss his theory as misguided and pursue our ontological inquiry as before.

Alternatively, we can take Merker's theory seriously and see where it leads us. Merker says his subject matter is "the state or condition presupposed by any experience whatsoever" (sect. 1, para. 2), or the "'medium' of any and all possible experience" (sect. 1, para. 3). He then gives us a detailed account of such a medium, couched in terms of neural systems, their functions, and their interrelations.

Insofar as philosophers talk about anything that sounds like this, it is what they sometimes call *creature consciousness*. For present purposes and to a first approximation, creature consciousness is whatever differentiates ordinary people who are either awake or in REM sleep from ordinary people who are in non-REM sleep, in a coma, and so forth. This seems to be what Merker is theorizing about.

When it comes to understanding phenomenal consciousness, many philosophers would maintain that creature consciousness is mostly irrelevant to the ontology of phenomenal consciousness. According to the philosophical mainstream, the ontological key to phenomenal consciousness resides in state consciousness.

Merker, however, says his subject matter is consciousness in its most "basic" sense. Perhaps he is onto something. Perhaps creature consciousness is at least partially constitutive of phenomenal consciousness. What would this mean? Most people agree that creature consciousness is a necessary condition for state consciousness. Perhaps there is more to creature consciousness than that.

From the point of view of neuroscience, creature consciousness is a global state of (part of) the brain – the difference between ordinary people's brain when they are awake or in REM sleep and their brain when they are in non-REM sleep, in a coma, and so forth. My suggestion is that creature consciousness thus understood contains at least part of the ontological basis of phenomenal consciousness. In other words, a (more or less large) part of what makes a system have experiences is that it is creature-conscious.

Under this view, state consciousness may be understood as follows: A state is state-conscious if and only if it is the state of (a spatio-temporal part of) a creature-conscious brain, or better, an appropriate kind of state of (a spatio-temporal part of) a creature-conscious brain. There remain, of course, two important questions. First, what is the difference between those states of creature-conscious beings that are phenomenally conscious and those that are not? Second, what else is needed (if anything), besides creature consciousness, for full-blown phenomenal consciousness? An adequate theory of consciousness would have to answer these questions.

What kind of global brain state corresponds to creature-consciousness? Is it physical, functional, representational, or a combination of these? According to Merker, creature consciousness is the product of an analog reality simulator that integrates sensations and motivations to select targets and actions. Perhaps his view could be glossed as follows: When the simulator is operating, the system is creature-conscious; when the simulator is idle (for whatever reason: rest, breakdown, etc.), the

system is creature-unconscious. Integrating sensory information and motivations as well as selecting targets and actions appear to be broadly functional and representational notions. So Merker appears to be offering a functional/representational account of creature consciousness.

There is at least one other option. Perhaps creature consciousness requires some special physical properties, analogously to the way water's power to dissolve certain substances and not others requires a certain molecular composition and molecular structure at a certain temperature (cf. Shapiro 2004). I cannot elaborate further. Differentiating clearly between physical, functional, and representational accounts of creature consciousness would require an adequate account of the distinction between the physical, the functional, and the representational, and there is no room for that here.

The present suggestion has epistemological consequences. If creature consciousness were at least partially constitutive of phenomenal consciousness, it would be a mistake to develop theories couched solely in terms of state consciousness, without saying anything about creature consciousness – as philosophers are fond of doing. Rather, a correct approach to phenomenal consciousness should begin with an account of creature consciousness.

Before concluding, it may be helpful to distinguish several different claims: (1) the brainstem is necessary to sustain and regulate creature consciousness (uncontroversial), (2) the brainstem can sustain creature consciousness by itself (Merker's theory), (3) the brainstem can be the locus of conscious experience (Merker's theory), and (4) creature consciousness is (at least part of) the ontological basis of conscious experience.

Thesis (3) is stronger than (2), and Merker does little to support (3) as opposed to (2). (Do children with hydranencephaly go into anything resembling REM sleep? Evidence that they do would support [3].) Perhaps he intends to make a further claim: (5) creature consciousness is sufficient for phenomenal consciousness. Thesis (5) is even stronger than (4). However, in light of unconscious cognition, including phenomena such as blindsight, (5) is hard to swallow without at least some qualification.

But we don't need to accept all of Merker's claims in order to consider (4). In fact, claim (4) can be motivated on the grounds of (2) or even (1) alone, and (1) is uncontroversial. If phenomenal consciousness can occur without a cortex, as Merker believes, then the challenge posed by (4) becomes more forceful and more difficult to avoid. But, regardless of the extent to which we agree to Merker's theory, we should consider the possibility that (4) is correct.

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Who dominates who in the dark basements of the brain?

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Abstract: Subcortical substrates for behavioural integration include the fore/midbrain nuclei of the basal ganglia and the hindbrain medial reticular formation. The midbrain superior colliculus requires basal ganglia disinhibition in order to generate orienting movements. The colliculus should therefore be seen as one of many competitors vying for control of the body's effector systems with the basal ganglia acting as the key arbiter.

Understanding the brain's functional architecture is certainly key to unlocking the mystery of the coherence of behaviour, and even, perhaps, consciousness. In this regard, Merker usefully draws our attention to subcortical systems as critical loci for behavioural integration that may instantiate some form of supra-cortical control. As we have previously argued (Prescott et al. 1999), combining Penfield's notion of a centrencephalic dimension to brain organization with a view of the brain as a layered control system provides a powerful set of explanatory concepts for understanding how the vertebrate brain architecture has adapted, with little change to its basic "groundplan," to many different body types and ecological niches. The particular set of brainstem substrates that Merker has chosen to emphasize seems, however, somewhat curious. The roles of the colliculus in orienting, the periaqueductal grey in behavioral patterning, and the hypothalamus in motivation are not controversial, but the promotion of the colliculus to the "functional apex" of processing for target selection is surprising, as is the suggestion of the zona incerta (ZI) as a key locus for action selection. In our view, other centres, either side (i.e., both more rostral and more caudal) of Merker's "selection triangle," may be more important in subserving these important aspects of behavioural integration.

One such group of structures are the basal ganglia (BG). This collection of fore- and mid-brain nuclei, identified by Thompson (1993) as a major component of the centrencephalic core, is located in such a way that its principal input structure (striatum) is rostral, and its output structure, substantia nigra (SNr), caudal to Merker's "synencephalic bottleneck." The BG are therefore ideally placed to provide the required funnel from distributed cortical processing to sequential brainstem operation. Merker discusses the functional role of the BG, primarily in relation to this "data reduction" context, as providing action-related information to the colliculus. However, the BG appear to be doing something more significant than simply providing the colliculus with one of its several sources of afferent input. Specifically, the tonic inhibition provided by the SNr maintains a veto over the capacity of the colliculus to generate orienting movements (Hikosaka et al. 2000). In the case of a visual stimulus, for example, this veto is only removed when there is sufficient excitatory input onto the oculomotor region of the striatum to cause inhibition of SNr and, thence, disinhibition of the collicular motor layer. The colliculus itself provides afferent input (via thalamus) to relevant striatal neurons that, together with convergent signals from cortex, the limbic system, and elsewhere, determine the significance of the stimulus (McHaffie et al. 2005). It is therefore the BG, not the colliculus, that sees the full gamut of pertinent, contextual information and is thus the dominant partner. Without BG gating, the colliculus would initiate orienting to any target that generated a strong, spatially localized phasic stimulus. The BG add intelligence to this reactive process by preventing orienting to high-amplitude but uninteresting stimuli, and enabling it to weaker, but potentially more significant, triggers. A broad range of empirical studies, theoretical proposals, and computational models (for reviews see Gurney et al. 2004; Redgrave et al. 1999) support the proposal that the BG operate as an action selection mechanism, not just for collicular control of orienting, but for competing sensorimotor systems throughout the brain. From this perspective, the colliculus is just one of many competitors vying for control of the body's effector mechanisms, with the BG as the key arbiter.

A remarkable feature of the BG is the homogeneity of their intrinsic circuitry. This observation adds weight to the hypothesis that these nuclei implement a consistent function despite

the functional diversity of brain areas to which they interface. In contrast, the ZI, highlighted by Merker as a possible action selection locus, is a very heterogeneous structure (Mitrofanis 2005). Furthermore, evidence from functional studies suggests other possible modulating roles: for instance, Trageser et al. (2006) reported ZI's involvement in gating ascending sensory inputs according to the animal's current state of arousal.

Although the BG instantiate a dominant integrative centre in the intact adult brain, studies of infant and decerebrate rats suggest the presence of an alternative locus for action integration further down the neuraxis. A possible candidate, first suggested by the Scheibels (1967), is the medial core of the reticular formation (mRF). This hindbrain structure receives input from many cortical and subcortical brain systems and directs its output to movement generators in the brainstem and spinal cord. We recently sought to promote interest in the mRF by elucidating its anatomy (Humphries et al. 2006), and by developing new simulation and robotic models of this structure viewed as an action selection mechanism (Humphries et al., in press). The mRF is organized as a set of linearly arranged cell clusters, likened by the Scheibels to a "stack of poker chips." In Humphries et al. (in press) we proposed, and demonstrated in simulation, that activity in individual clusters may represent *sub-actions* – component parts of a complete behavior. Effective control by the mRF would therefore involve simultaneous activation of clusters representing compatible subactions and inhibition of clusters representing incompatible ones. The mRF is a major target of BG output (via the pedunculopontine nucleus) and, in the intact adult brain, both systems are likely to cooperate in determining what behaviour is expressed at a given time. The relationship between the two systems may combine aspects of layered and hierarchical decomposition of control. Layered, because developmental and lesion studies suggest that the mRF can operate, to some degree, without modulation from higher brain structures (including BG). Hierarchical, because patterns of mRF coordinated behavior could be selected *in toto* by BG focal disinhibition.

For Wilson (1925), the BG, lying towards the base of the brain, had "the characteristic of all basements, i.e. darkness." Although many windows have been opened onto BG function since Wilson's era, other subcortical nuclei still reside in subterranean obscurity. Despite the gaps in our knowledge, Merker is right to try to discern some structure amidst the gloom. With regard to his specific hypotheses, however, there is no compelling reason for viewing the ZI as the central arbiter, or the colliculus as the target selector. In the dark basements of the brain the basal ganglia dominate both.

Should the superficial superior colliculus be part of Merker's mesodiencephalic system?

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Abstract: The superficial superior colliculus appears to be a primitive visual analyzer whose function has been taken over by the visual cortex, most completely in man. The phenomenon of blindsight shows that, although intact, the superior colliculus cannot by itself provide conscious perception in human patients. Is it possible that, in anencephalic children, it recovers the role it had in lower mammals?

Nowadays, we tend to believe that all brain functions are localized. The reason is that modern techniques – from unit recordings in behaving animals to fMRI in humans – are geared to finding functions localized. It can hardly be otherwise, because data obtained by these techniques are publishable only if they lead to the discovery that the particular behavior or function under study can be attributed to a given structure or type of neurons. If the research finds no such evidence, the data will not be published. Thus is introduced an obvious bias in our views, and it is unfortunate because some functions may depend more on circuits of interactions between different structures than on the activation of neurons in any one of these structures particularly. The target article by Björn Merker adopts another approach. It is a refreshing effort of integration. The problem of consciousness is among the most difficult, because consciousness is so hard to define, difficult to test, and it seems to depend on the integrity of a number of functions (albeit none of them absolutely essential in all situations), such as memory, perception, attention, emotional concern, language, and other motor behaviors – even laughing. Indeed, laughing happens to be one of the criteria that Merker uses in evaluating the conscious state of anencephalic children.

I think that Merker is right in pointing out that consciousness is neither obviously nor necessarily a cortical function. His anecdotal observations of anencephalic children are impressive and important, both from scientific and ethical viewpoints. Merker stresses the role of a mesodiencephalic group of structures and I agree on this idea, but I am a little surprised to see included in this group the superior colliculus or, at least, its superficial layers.

The most superficial layers of the superior colliculus are essentially visual. Their organization is certainly more primitive than that of primary visual cortex, but still, it is topographic. It is probably relevant to consider the phenomenon of blindsight (Weiskrantz et al. 1974) in discussing the possible participation of the superior colliculus in consciousness. Patients who have a circumscribed lesion of their primary visual cortex are blind in the corresponding region of their contralateral visual field. They say that they don't perceive a visual stimulus presented in that region. Yet, when forced to do so, they can report the presence or absence of such a stimulus with surprising accuracy. Even features like size or orientation often are "guessed" correctly. The phenomenon of blindsight suggests a couple of remarks.

First, because conscious perception is lost in blindsight but the superior colliculus is intact, it is difficult to argue that the latter plays a major role in consciousness in an adult human brain. Maybe, in an anencephalic child, the superior colliculus has recovered the function of visual analyzer that has been transferred to the cerebral cortex during the course of evolution.

Second, as visual discrimination is spared – at least partly – but conscious perception is lost in human blindsight, it also seems difficult to take the persistence of visual discrimination after brain lesion in primates as evidence relevant to consciousness. Visual discrimination can exist without consciousness.

The superior colliculus has been one of the most thoroughly investigated structures in rodents (hamsters and rats), in cat, and in monkey, in whom it plays a significant role in vision. In these species, the visual physiology of the upper layers of SC has been as abundantly studied as that of visual area V1. But, remarkably, we have much less information about the significance (if any) of the superior colliculus in man. There is remarkably little known pathology of the superior colliculus (e.g., in contrast to more ventral structures such as the interstitial nucleus of Cajal, the medial longitudinal fasciculus, the red nucleus, and reticular formation). It is quite conceivable, in fact, that the human superficial superior colliculus is no more than a remnant of an ancient visual analyzer. In contrast, the deeper superior colliculus is structurally more like the adjacent mesencephalic reticular

formation and could, indeed, be a part of the system described by Merker.

The functional utility of consciousness depends on content as well as on state

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Abstract: This commentary considers Merker's mesodiencephalic proposal in relation to quantitative measures of neural dynamics suggested to be relevant to consciousness. I suggest that even if critical neural mechanisms turn out to be subcortical, the functional utility of consciousness will depend on the rich conscious contents generated by continuous interaction of such mechanisms with a thalamocortical envelope.

Merker's target article provides a lucid and compelling alternative to currently dominant (thalamo-)corticocentric proposals regarding the loci of neural mechanisms underlying consciousness. Taking a quantitative perspective, this commentary challenges Merker's claim that the functional utility of consciousness is independent of the level of sophistication at which conscious contents are integrated. I also comment on the proposed function of consciousness in the coordination of motivation, action, and target selection, and finally, I suggest some implications for nonhuman consciousness.

An important step in the evolution of scientific theory is the development of useful quantitative measures that connect different levels of description. The scientific study of consciousness requires such measures in order to generate explanatory links between features of neural activity and features of phenomenal experience. Several recent studies have discussed various measures of the "dynamical complexity" of neural activity, including "neural complexity" (Edelman & Tononi 2000; Tononi & Edelman 1998), "information integration" (Tononi 2004), and "causal density" (Seth 2005; Seth et al. 2006). These measures share the idea that the dynamical complexity of a neural system reflects the extent to which the activity of its components is both *differentiated* (i.e., small subsets of a system are relatively independent of each other) and at the same time *integrated* (i.e., large subsets tend to behave coherently).

Critically for theories of consciousness, the balance between differentiation and integration is also a fundamental aspect of phenomenal experience: Each conscious scene is one among a vast repertoire of possible conscious scenes (differentiation) and yet is experienced as a unified whole (integration) (Tononi & Edelman 1998). Therefore, a well-specified measure of dynamical complexity can provide an explanatory link between neural activity and phenomenal experience. Importantly, cortical networks appear particularly well suited to generating neural dynamics of high complexity (Sporns et al. 2000).

The detailed description of mesodiencephalic mechanisms provided by Merker raises the interesting possibility that mesodiencephalic and corticocentric models could be compared on their propensity to generate complex neural dynamics. Although such modeling work remains to be done, it seems plausible that a model mesodiencephalon by itself would *not* support neural activity of high dynamical complexity, at least when compared to a model thalamocortical system. Why? Previous computational models of closely associated mechanisms that are also involved in sensorimotor selection, such as the basal ganglia and the medial reticular formation, reveal dynamical properties appropriate for segregation of multiple competing sensorimotor streams (Humphries et al., in press; Prescott et al. 1999). Such dynamical segregation seems inconsistent with the integration required for high values of complexity. Moreover, the small size of mesodiencephalic systems as compared to thalamocortical systems, in terms of numbers of neuronal elements, suggests that the latter should support dynamics with greater differentiation.

Having dynamics of high complexity is important not only in accounting for fundamental aspects of phenomenology, but also for supplying functional utility. According to the "dynamic core hypothesis" of Edelman and Tononi (2000) and its recent extensions (Edelman 2003; Seth et al. 2006), the functional utility of a complex neural/phenomenal state is that it provides a highly informative *discrimination*. By being differentiated, any given conscious state is distinct from an enormous repertoire of other states, each reflecting different combinations of internal and external signals. By being integrated, each conscious state can appear as distinct *to the system itself*, and is therefore useful *for* the system in guiding action.

This position differs from Merker's claim that the functional utility of consciousness "will turn out to be independent of the level of sophistication at which the contents it integrates are defined" (sect. 1, para. 6). From the point of view of discrimination, functional utility will correlate closely with the sophistication of conscious contents. A richly elaborated conscious scene will provide a more informative and hence a more useful discrimination than a comparatively impoverished scene. In other words, the functional utility of consciousness should not be construed only in terms of conscious "state" (i.e., a position on a continuum ranging from coma to normal alert wakefulness), independent of the degree of elaboration of conscious "content" (i.e., the richly differentiated components of each conscious experience). As Merker makes clear, subcortical mechanisms are proposed as a locus for the generation of conscious state, whereas conscious contents remain dependent on cortex. Thus, even if critical neural substrates turn out to be subcortical, the functional utility of consciousness will depend on cortical systems, as well.

Merker himself argues that consciousness is useful for integrating target selection, motivational modulation, and action selection. This proposal marks a valuable departure from many previous studies, which, possibly for reasons of practical necessity and misplaced conceptual hygiene, treated these overlapping and interdependent processes as being in principle separable and independent (see Seth [in press] for further discussion of this issue). Merker's proposal can also be viewed in terms of discrimination, because each integration can be thought of as being an informative discrimination among a repertoire of motivationally modulated sensorimotor mappings. Moreover, that such integrations are suggested by Merker to take place in a conscious "analog reality space" parallels the dynamic core hypothesis in proposing that conscious qualia *are* high-order discriminations in a multidimensional signal space (Edelman 2003).

Finally, it is worth considering the important question of nonhuman consciousness. A strong case can be made that the ability of organisms to verbally report conscious contents should *not* be taken as a necessary criterion for consciousness (Seth et al. 2005). Rather, by using humans as a benchmark, a number of interlocking criteria can be identified, at both behavioral and neurophysiological levels of description. These criteria include "informativeness" as measured by dynamical complexity. Whereas in humans and other mammals the relevant dynamical complexity may depend on the interaction of a mesodiencephalic system with a thalamocortical system, in non-mammals it may depend on different anatomies, for example, a differentiated telencephalon in birds, and the optic, and vertical, and superior lobes in cephalopods (Edelman et al. 2005). In any case, by shifting the theoretical spotlight away from cortex and towards architectonic features that are conserved among a wider range of species, Merker's article lies squarely in the productive tradition of challenging human and mammalian privilege.

Raw feeling: A model for affective consciousness

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Abstract: Seeking to unlock the secrets of consciousness, neuroscientists have been studying neural correlates of sensory awareness, such as meaningless randomly moving dots. But in the natural world of species' survival, "raw feelings" mediate conscious adaptive responses. Merker connects the brainstem with vigilance, orientating, and emotional consciousness. However, depending on the brain's phylogenetic level, raw feeling takes particular forms.

Philosophical debate on consciousness is ageless, but detailed neurobiological models are a recent development. One of the best among the latter is global workspace theory (GWT; Baars 1988); which subscribes to the traditional definition of consciousness as subjective awareness of momentary experience interpreted in the context of memorized past and expected future. Consciousness in the "cortico-centric" GWT is conceived of as transient synchronized thalamo-cortico-cortical neural activity.

The GWT-like framework of Crick and Koch (1998; 2003), attempts to reduce consciousness to measurable properties by explicitly leaving out emotions and feelings. But what remains in such accounts of consciousness? Conversely, a growing body of theory maintains that the study of consciousness and emotion will yield new insights (Damasio 1999; Greenfield 2000). The theoretical analysis of Merker, supported by his notable findings in hydranencephalic children, adds important impetus to this movement.

Consciousness and emotion. Fundamental insights have been gained by studying "purely cognitive" processing, but virtually all conscious experience carries an affective tone (Ashton 2002). This affective tone, designated as "raw feeling" (Panksepp & Panksepp 2000) influences information processing faculties such as attention, memory, and decision-making, which have been associated with consciousness in both traditional and contemporary theories (Baars 1988; Damasio 1999).

Panksepp and Panksepp (2000) broke the boundaries of traditional theories of consciousness by proposing a double-layered model wherein a secondary cortico-centered form supervenes on a subcortico-centered primary form of consciousness. According to Panksepp and Panksepp (2000), brain evolution shows that the secondary cognitive forms of consciousness emerged from the primary affective forms. Moreover, they argue that our "raw emotional experiences" are created subcortically and constitute the primordial neural ground upon which all forms of conscious processing are built. Emotions, therefore, do not merely provide for "global valence tagging" in the cognitive realm, but mediate the subject's strategic quest for adaptive homeostasis in both immediate (e.g., hunger, thirst, fear, anger) and more enduring timeframes (e.g., goal-directed behavior, dominance status, attachment/bonding) (Schutter & Van Honk 2004a; Van Honk & Schutter 2005). In the next subsection, a triple-layered model of "affective consciousness" adapted from Panksepp and Panksepp (2000) is outlined. It might serve the psychobiological investigation of embodied awareness in a manner consistent with the compelling hydranencephalic evidence amassed by Merker against the exclusively cortical model of consciousness.

A model of affective consciousness. Consciousness evolved to ensure adaptive homeostasis (Damasio 1999; Panksepp & Panksepp 2000; Schutter & Van Honk 2004b). The mechanism relies on the subject's capacity to experience raw feelings of reward and punishment, which evoke functional behavioral responses. This core feature works together with the ability to detect (on basis of motivated attention) and to evaluate (on basis of instinct/emotional memory) rewards and punishments and to make fine-tuned decisions of approach or withdrawal-related action (Ressler 2004; Schutter & Van Honk 2004b). Reminiscent of the triune brain theory of Paul MacLean (1990), we propose a theoretical framework which encompasses three detection-evaluation-decision (DED) devices that mirror phylogenesis observed in the instinctual reptilian, emotional paleomammalian, and cognitive neomammalian brain (cf. Panksepp 2005a for a related but more strongly bottom-up regulated 3-level model). These DED devices are concordantly instinctual, emotional, and cognitive in nature, but their working is also orchestrated by raw feelings that, depending on the level, come as instinctual drives, emotional biases, and cognitively guided mood states.

On the different phylogenetic levels there are structural convergence zones wherein core brain areas influence the content of affective consciousness. In the reptilian brain, DED processing occurs at an instinctual brainstem level. For example, on its most primitive level, the vagus reflexively copes with threat by way of immobilization behaviors such as passive avoidance (Porges 2001). Crucially, there is evidence showing that parasympathetically mediated immobilization behaviors are mediated by raw feelings in the form of instinctual drives (e.g., Hofer 1994). Thus, primordial DED processing at the level of the vagus nerve is instinctual, implicit, and therefore of a non-cognitive nature.

In the paleomammalian or emotional brain, the DED system copes with threat by initiating flight/fight behaviors that are modulated by neuroendocrine mechanisms at the level of the amygdala and hypothalamus (Van Honk & Schutter 2005). The involvement of the amygdala in different aspects of affective processing is especially well documented. This small medial temporal lobe structure has extensive connections with all major subcortical and cortical structures involved in motivation, emotion, and emotion regulation. Receiving information indirectly from the sensory cortices and directly from the thalamus, the amygdala participates in both implicit and explicit forms of DED processing (Davis & Whalen 2001; LeDoux 2002). Orchestrated by raw feelings in the form of emotions, the amygdala DED mechanism copes with threat by initiating flight-fight behaviors.

The neomammalian-cognitive brain possesses our higher-order cognitive faculties such as reasoning and language (Damasio 1994). Affective consciousness is not rooted here but can be accessed and modulated in a top-down fashion (Block 1995). A brain structure importantly involved in cognitive emotional DED processing is the orbitofrontal cortex (OFC) (Rolls 1999), which is highly interconnected with other cortical and subcortical brain areas. At the level of the OFC, motivated behavior is explicit, cognitively controlled, and effortful in nature. Behavior also carries social features, and the arsenal of responses to challenges employed by DED include instrumental acts wherein complex emotion-cognition interactions take place. All of these are directed by raw feelings in the form of cognitively laden mood states.

These are the core principles of our perspective on affective consciousness, a triple-layered instinctual-emotional-cognitive adaptation that follows the phylogeny and ontogeny of brain development and wherein reverberating neurodynamic affective maps are continuously created at the brain's phylogenetic levels. These affective maps constitute raw feelings on different processing levels in the brain – a triple balance supporting global adaptive homeostasis bound into a unitary experience. However, drawing upon Jackson's (1958) principle of dissolution, MacLean's (1990) notion of loosely coupled systems, and the polyvagal theory of Porges (2001), important insights can be

gained into consciousness by scrutinizing evolutionarily separate functions on behavioral and physiological levels. Merker's story provides some of these insights and may contribute importantly to theories on the "what and where" of consciousness.

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The human superior colliculus: Neither necessary, nor sufficient for consciousness?

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Abstract: Non-invasive neuroimaging in humans permits direct investigation of the potential role for mesodiencephalic structures in consciousness. Activity in the superior colliculus can be correlated with the contents of consciousness, but it can be also identified for stimuli of which the subject is unaware; and consciousness of some types of visual stimuli may not require the superior colliculus.

Merker presents a wide-ranging overview in which a central role for the mesodiencephalic system in consciousness is proposed. Specifically, it is suggested that activity in the superior colliculus (SC) is necessary for changes in conscious content to occur, and activity in mesodiencephalic structures is sufficient to support consciousness. In humans, there is increasing evidence that activity in subcortical structures, such as the SC, can indeed be correlated with the contents of consciousness. Human SC is visually responsive in a retinotopic fashion (Schneider & Kastner 2005; Sylvester et al. 2007), and Merker highlights our recent demonstration that changes in SC activity (accompanied by similar changes in activity in retinotopic early visual cortex) are correlated with altered perception in a visual illusion induced by sound (Watkins et al. 2006). Moreover, other subcortical structures anatomically adjacent and closely linked to the SC, such as the lateral geniculate nucleus, show fluctuations in activity closely correlated with changes in the contents of consciousness during binocular rivalry (Haynes et al. 2005; Wunderlich et al. 2005). But after damage to human primary visual cortex, SC activity can also be observed when moving visual stimuli are presented in a blind hemifield (Sahraie et al. 1997). Moreover, such SC activation can correlate with the emotional content of faces again presented in the blind hemifield (Morris et al. 2001). Such processing of subjectively invisible visual stimuli associated with SC activation can be associated with residual visual sensitivity (or "blindsight"; Weiskrantz 1997), which in turn may be related to different patterns of SC connectivity in patients with blindsight following hemispherectomy (Leh et al. 2006). Taken together, these data suggest that activation of the superior colliculus alone is therefore not *sufficient* for awareness, at least after damage to primary visual cortex.

The notion that activity in mesodiencephalic structures alone is insufficient to support consciousness is challenged by Merker's fascinating personal observations of the behavior of children with hydranencephaly. Despite these children apparently lacking most functioning cortical structures, a range of behaviors is reported that indicates some degree of limited responsiveness to their surroundings. However, caution is required before concluding that these individuals are conscious, and indeed, interpreting this as reflecting preserved mesodiencephalic function. Hydranencephaly describes a range of brain malformations that may vary with respect to time of onset, pathogenesis, and organization of any cortical remnants that may be present (Halsey 1987); and survival beyond six months is rare (McAbee et al. 2000). In the presently reported cases, the extent of cortical damage is unclear, so the extent to which any behaviors reflect mesodiencephalic structures alone in these individuals is not known. Moreover, responsiveness to the environment is a capacity exhibited by nearly any organism with a central nervous system, and cannot be unambiguously taken as a marker of consciousness. Verbal or manual reports are generally considered the primary criterion that can establish whether a percept is conscious (Weiskrantz 1997). Such behaviors, demonstrating intentionality, are not clearly evident in the present observations and many of the reported behaviors could be generated unconsciously or reflexively. This emphasizes both the difficulty in determining whether an individual unable or unwilling to give verbal or manual reports is conscious (Owen et al. 2006), and the consequent need to explore the possibility that non-invasive biomarkers of consciousness might be developed to permit such inference.

Three indirect lines of evidence also suggest that SC activation in humans may not be necessary, either, for changes in the contents of consciousness to occur. First, visual stimuli that stimulate only short-wave-sensitive cones (S-cones) in the retina are clearly visible (and indeed can influence attention and behavior; Sumner et al. 2006), even though the SC receives no direct projections from short-wave-sensitive cones and is therefore unlikely to be activated by such stimuli. Second, although SC damage in humans can cause lateralized visual neglect (Sprague 1996) and consequent failure to represent the contents of consciousness in one half of the space, bilateral damage does not eliminate awareness (Weddell 2004). Finally, direct intracranial stimulation of human visual cortex that bypasses geniculostriate and retinotectal pathways can result in conscious visual percepts (Lee et al. 2000), suggesting that subcortical activity may not be necessary for all types of awareness. Although all these lines of evidence are indirect, they raise the question of whether SC activity is strictly necessary for all types of conscious visual percept.

The picture that emerges, at least in humans, appears to be more complex than a simple identification of particular parts of the mesodiencephalic system with a single role as a necessary and sufficient "gatekeeper" for the contents of consciousness. Indeed, it seems unlikely that activity in any single area of the human brain will be sufficient for consciousness (Rees et al. 2002). The consistent association of changes in activity in SC (and other subcortical) structures with fluctuations in awareness thus suggests that they may play a role as part of a network of cortical and subcortical areas whose activity might represent a minimally sufficient substrate for the contents of consciousness; but further research is required.

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Affirmative-action for the brainstem in the neuroscience of consciousness: The zeitgeist of the brainstem as a "dumb arousal" system

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Abstract: Merker offers a remarkable statement about the neural integration essential to conscious states provided by the mesodiencephalon. The model for triangular interaction between action selection, target selection, and emotion is heuristic. Unfortunately, there is little interest (relatively speaking) in neuroscience in the mesodiencephalon, and attention is currently heavily directed to the telencephalon. This suggests that there may be less real momentum than commonly assumed towards the Holy Grail of neuroscience, a scientific theory of mind, despite the major upsurge in interest.

It is a great privilege to comment on such a remarkable and brilliantly integrative essay. Although bits and pieces of this argument have been in the literature in various forms for a while, the full and extended summation of them in the target article is original, and at the same time, timely and badly needed. It is urgently needed at a time when the equation of consciousness with cortical function, if anything, is only deepening in neuroscience, particularly within cognitive neuroscience, where functional imaging study after study generates images showing (primarily, albeit not exclusively) cortical activation.

Neuroscience still fundamentally lacks its keystone, a validated theory of consciousness. I agree strongly with Merker that until we understand the complexity of the deep integrations taking place within many dozens of brainstem structures and then their interdigitation with thalamus and cortex, we will move no closer to the Holy Grail of neuroscience – that is, a neural theory of mind. That consciousness must rest in some form of neurodynamic integration seems the only certainty. That it might be marked in cortex by higher frequency oscillations putatively linking distributed cortical regions does not help us understand what the requisite and essential neurodynamics of the upper brainstem might be. Only the superior colliculus (SC) appears to follow the gamma and beta oscillatory pathways of cortex.

Merker's article starts with the central heuristic that consciousness is a way of matching needs with opportunities as part of a centralized interface for action and target selection. He describes consciousness as arising out of a "motion-stabilized body–world interface" (sect. 7), presenting potential targets for action, while motivational systems "bid" competitively into that interface to both select targets and also to select actions. I believe he is correct that consciousness must bring together target selection, action selection, and motivation to optimize integration for action in real time, with the integration highly adaptive and selected on this basis. In other words, consciousness may *emerge from* interdigitation of attention, action selection, and emotion/homeostasis. These concepts are very similar to those I independently presented in a previous publication with a colleague, and in an ASSC (Association for the Scientific Study of Consciousness) electronic seminar[1] (Watt & Pincus 2004; Watt 1998). Jaak Panksepp has also separately suggested that consciousness is dependent on the integration of sensory maps, motor maps, and homeostatic/affective information (Panksepp 1998a), and Damasio has proposed somewhat similar notions (1999). The issue here is not "who came up with the idea first," but rather that *different theorists and researchers are coming to essentially the same conclusion quite independently of one another*. Thus, despite the chaos in the neuroscience of consciousness, a broad-based confluence of ideas is forming in a still inchoate form.

To create any kind of theory of conscious state without first considering how the brain might integrate sensory processing, and motor processing with emotional/homeostatic processing seems a doomed venture. The phenomenological/behavioral priority of experiences such as hunger and pain argue that homeostasis has "ground floor" involvement in the machinery of consciousness, consistent with selection mandating that consciousness promote survival by *prioritizing* homeostasis. As the simplest and most basic paradigm for consciousness, sensory systems mapping an image of food, motor systems mapping trajectories to the food, and a homeostatic representation of metabolic shortfall must be in register with one another in order for an organism to do something as simple as eat when energy is low, in the presence of food. I suspect this integration of motor and sensory and homeostatic operators may not only be taking place between the colliculi, periaqueductal gray (PAG), and motor systems in the brainstem as outlined by Merker, but also within the "extended reticular thalamic activating system" (Newman & Baars 1993) Therefore, it might be more accurate to characterize these as "smart integration systems" rather than as "dumb arousal systems."

This notion of the reticular brainstem as a "dumb arousal system" is complementary to the assumption that "consciousness is in the cortex." The concept of a dumb arousal system suggests that the brainstem does for the forebrain essentially what a battery does for a light. This "dumb arousal" concept is a begrudging acknowledgment of the original work by Moruzzi and Magoun (1949) on the reticular activating system, but it is far less than the system-wide functional integration that Merker argues is the real contribution of the mesodiencephalon. The "dumb arousal" concept (in my judgment) may have actually set back (more than we appreciate) a truer functional understanding of the brainstem and indeed of consciousness itself. The "dumb arousal" concept also generated a naïve optimism that we could compensate for brainstem injuries that caused severe disorders of consciousness through brainstem or thalamic electrical stimulation therapies. By and large, these have been spectacularly unsuccessful. Perhaps we are missing something. Certainly such a simple concept could do little justice to the functional complexity of the brainstem, which contains 40+ nuclei, with a staggering diversity of connections, neuromodulators, and functional correlates.

A question rarely asked about this concept for the reticular brainstem as a "nonspecific arousal system" is, "what does this *really* mean?" First of all, the notion of arousal as being "nonspecific" is clearly mistaken from the standpoint of widely differential contributions from these many reticular activating system structures. Additionally, the notion of "arousal" itself has been used in several different ways: (1) any process that increases firing rates of distributed forebrain neurons; (2) affective arousal (as in states of anger); and (3) global state shifts, such as into wakefulness, dreaming, and various stages of sleep. The first meaning (increased firing rates in forebrain) is not an adequate explanation at a neurodynamic level for the achievement of arousal in behavioral/affective terms, or for arousal to wakefulness, as consciousness cannot be meaningfully explained by the simple notion of "increased firing of forebrain neurons under brainstem influence." Lastly, arousal, as in simple arousal to wakefulness, is not a remotely adequate functional correlate for the extended RAS (reticular activating system), as wakefulness is preserved in PVS (persistent vegetative state), where no consciousness is present, often in the context of extensive RAS-mesodiencephalic lesions. Hence, "arousal to a conscious state" cannot be conflated with any kind of simple wakefulness, and requires other integrative functional "envelopes" (core/constitutive functions of attention, intention, and emotion). Therefore, if "arousal" simply means that *stimuli generate coherent behavioral responses (and signs of clear purposeful intent, emotion, and attentional tracking)*, this metaphor of "turning on the lights" begs crucial questions about how a vast array of brainstem structures (and their connectivities) might underpin creation of conscious states. In this sense, the assumed primary functional correlate ("nonspecific arousal") may be a non-explanation. *If the extended group of reticular systems enables coherent and purposeful behaviors to emerge from the system, then it cannot simply be "turning on the lights" but must be underpinning a global integration of function of the kind Merker outlines.* The scientific challenge is now to map out this process, instead of being comfortably ensconced in an ignorance of which we are largely unaware. Without

more discomfort about that ignorance, we will fail to explore these questions adequately. Despite impressive gains, we know far less than we think we do.

NOTE

1. Email author for reprint of this article.

Author's Response

Grounding consciousness: The mesodiencephalon as thalamocortical base

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Abstract: My response addresses general commentary themes such as my neglect of the forebrain contribution to human consciousness, the bearing of blindsight on consciousness theory, the definition of wakefulness, the significance of emotion and pain perception for consciousness theory, and concerns regarding remnant cortex in children with hydranencephaly. Further specific topics, such as phenomenal and phylogenetic aspects of mesodiencephalic-thalamocortical relations, are also discussed.

It was with some trepidation that I turned to the many commentaries on my target article, but the constructive tenor of collegial exchange and criticism that met me in their pages sustained me through my work on this response. Six issues recurred with sufficient frequency to merit general treatment, namely, my neglect of the forebrain contribution to human consciousness, the implications of so-called blindsight for consciousness theory, questions related to the definition and mechanisms of wakefulness, the nature of emotion and its subcortical organization, the significance of pain perception for consciousness theory, and concerns regarding remnant cortex in children with hydranencephaly. I will deal in general terms with each of these in turn before attending to additional issues on an individual basis.

R1. My deliberate neglect of the telencephalon

Considering the set of commentaries as a whole, no single issue appears to have caused more problems than my attempt to leave the forebrain on the sidelines while exploring whether any kind of phenomenal consciousness might, in fact, be implemented at brainstem levels in the absence of or without reliance upon telencephalic mechanisms. In different ways and to varying extent, a number of suggestions, questions, or objections contained in the commentaries by **Aboitiz, López-Calderón, & López** (**Aboitiz et al.**), **Barcelό & Knight**, **Behrendt**, **Coenen**, **Collerton & Perry**, **Edelman**, **Freeman**, **Gilissen**, **Morin**, **Morsella & Bargh**, **Seth**, and **Watkins & Rees** concern my neglect of the obvious and massive contribution of the telencephalon to adult human consciousness. Let me assure these commentators that I harbor no greater doubts than they do about its importance in this regard. However, the topic clearly announced in the title of my target article is not that of accounting for adult human consciousness, but whether a conscious mode of function is conceivable apart from cortical mechanisms. In order to explore that question, I set out to search for grounds upon which some form of phenomenal consciousness might prove to have functional utility at more basic levels of neural organization, and if so, to try to identify neural mechanisms at the level of the brainstem that might plausibly implement such a mode of conscious function.

I found those grounds in the enhanced control economy, which I suggest can be achieved on the basis of interfacing target selection, action selection, and the ranking of needs in what I call a "selection triangle." I went on to propose that the triad of large structures physically encircling the brainstem reticular formation at the level of the midbrain, namely, the periaqueductal gray matter, the superior colliculus, and the substantia nigra (or their non-mammalian homologs/analogs), implements a vertebrate selection triangle, most particularly through a direct mutual interface of those three major midbrain components in the intermediate-to-deep (premotor, output-oriented) layers of the superior colliculus (Fig. 4 of the target article), layers which, in turn, project to the reticular formation. I suggested, moreover, that the format in which that interface is organized amounts to a conscious mode of function.

Needless to say, the phenomenal aspects of a candidate mode of conscious function implemented at that level would lack innumerable characteristics of adult human consciousness. I suggest, for example, that the "world" of its target selection domain would be devoid of three-dimensional objects, consisting instead of "a two-dimensional screen-like map of spatial directions on which potential targets might appear as mere loci of motion in an otherwise featureless noise field" (sect. 4.2, para. 10). I even suggest a concrete instantiation of such a phenomenology in the synthetic stimulus generated by Stoerig and Barth (2001). Such a visual world might appear threadbare to an adult human surrounded by the three-dimensional world supplied by his or her forebrain visual system, but it would be a visual world nevertheless. Moreover, I sketch a reason for why, even prior to forebrain expansion, *conscious* access to such a simple world might be preferable to dwelling in the dark night of unconsciousness – namely, as a means to implement the selection triangle in the form of an analog neural reality simulator – and how a system yielding such access might be structured.

My claim that the nested format proposed for the reality simulator amounts to *a conscious mode of function* (sects. 4.2 and 4.3) obviously reaches into the depths of definitional matters pertaining to consciousness, and accordingly, is unlikely to be settled in the short term. Suffice it to say that it accords well with the most global outlines of our own sensory consciousness, from whose implicit ego-center inside our body we gaze out at our world – a world that remains imperturbably stable, despite the body-based mobility of the receptor arrays which are our sole source of information about a physical universe. The key to understanding my entire proposal is this very claim, namely, that a neural arrangement which nests a body map within a world map around the origin of

a shared coordinate system (all three together serving economy of orienting for the fulfillment of needs) is conscious by virtue of that very arrangement itself, irrespective of its level of cognitive elaboration. This is because such an arrangement places a "subject" (the implicit perceptual ego-center under the influence of motivational bias)[1] in the presence of something other than itself (body and world, however primitively implemented), and accordingly, supplies the inherently perspectival (and asymmetric) relation which I believe supplies the principal diagnostic criterion for consciousness as such (Merker 1997).

I do not expect this claim to be taken at face value by just stating it, but it is essential to realize that the question posed in my target article is not "what is the neural organization of adult human consciousness?" but rather "might a lamprey conceivably be conscious, and if so, what might this imply for the neural organization of consciousness more generally?" Considering that, in ultimate terms, the only consciousness for which we can ever have direct evidence is our own, individual one, all such questions must of necessity be approached on indirect, circumstantial grounds. My target article accordingly arrays a highly diverse set of findings and arguments drawn from a range of disciplines spanning from comparative neurology, to behavioral neuroscience, to clinical neurology in order to sketch the outlines of at least one conceivable, if still tentative, affirmative answer to the lamprey question, and to educe some of its consequences for our conception of the neural organization of consciousness more generally.

Such a bid obviously does not amount to an account of human consciousness in full flower, let alone to a claim that its contents might "fit inside the midbrain," as it were. My target article elaborates on the far more limited aim just sketched, and by pursuing it I can hardly be faulted for neglecting the forebrain contribution to consciousness and the mechanisms that underwrite it, however important they may be in the final analysis. I have not even committed myself in the target article to an answer to the interesting and weighty question raised in the commentary by **Doesburg & Ward**: namely, to what extent, if any, the putative phenomenal content supported by an upper brainstem mechanism along the lines I sketch might, in fact, form part of the contents of normal adult human consciousness (see my response to their commentary, further on). All I have ventured to suggest is that in the absence of a cerebral cortex, and upon its prenatal loss more specifically, the brainstem might be capable of supporting a form of phenomenal consciousness on the basis of its own highly conserved and sophisticated sensory-motor-motivational circuitry.

R2. Blindsight, consciousness, and self-report

The approach just sketched would nevertheless be pointless if it could be shown that the very possibility of phenomenal consciousness were, in principle, abolished in the absence of all or some part of the telencephalic machinery. The much debated issue of how so-called blindsight might bear on consciousness theory has been interpreted by some to do just that, and because the issue was mentioned in this sense in commentaries by **Behrendt**, **Glassmann**, **Piccinini**, **Watkins & Rees**, **Schlag**, and **Doesburg & Ward**, a general comment is in order.

Blindsight refers to phenomena such as visually guided reaching and low-level visual discriminatory capacity exhibited by patients with cortical blindness caused by damage to their geniculostriate visual system. It should be clearly recognized that no mystery is attached to the basic fact that visual information may control behavior in the absence of a geniculostriate system: a number of visual systems complete paths from the retina to motor control subcortically, and others – notably the tectopulvinar system – do so by traversing extra-striate cortical paths (Goodale 1996; Ingle 1991; Weller 1988). The issue of blindsight in consciousness theory concerns which of these systems might support visual awareness and which ones do not.

If the very possibility of visual awareness were to be abolished by striate cortex lesions, then primary visual cortex would be necessary for visual consciousness, and this by extension would support a corticocentric model for consciousness more generally (though even then Sprague-effect type phenomena may complicate matters; see Pöppel and Richards 1974). This issue is controversial, and has been repeatedly reviewed (Cowey 2004; Pollen 2003; Tong 2003). We need not, however, enter into its details, because a crucial set of findings on the star patient of the blindsight research, known as GY in the literature, has radically recast the bearing of these phenomena on consciousness theory. Studies on this patient account for a disproportionate share of the blindsight literature, and for years he maintained that although he was aware of "something" during stimulus presentation in his affected visual field, it did not have the character of a visual percept. However, by asking him to match this "something" to synthetic stimuli presented in his good visual field, it has now been shown that his percept nevertheless is a distinctly visual one (Stoerig & Barth 2001).

In this patient at least, a destructive lesion of primary visual cortex has not eliminated the possibility of phenomenal visual consciousness in the affected parts of his visual field. Thus, until the blindsight phenomenon has been systematically subjected to the "matching" test, the presumption should be that blindsight phenomena harbor no radical implications for consciousness theory. There is a further lesson for consciousness research in this development: The availability of verbal self-report in humans has been regarded as a fundamental tool and asset of consciousness research, yet here is a clear instance in which reliance upon it has vitiated the inferences drawn from exacting laboratory studies. Verbal self-report by no means provides a "gold-standard" for determining the presence or absence of awareness (a point also made by **Anand**), particularly so in the many interesting circumstances in which potential contents of consciousness are marginal, unfamiliar for a variety of reasons, degraded, or near threshold.

It would be most natural and understandable if what GY meant by a visual percept were something like a "visual object," an instance of the fully formed three-dimensional object perception for which the cortical visual system evolved, but which, of course, is not the only kind of visual experience possible. The sample of the synthetic

stimulus accompanying the Stoerig and Barth report is most illuminating in this regard, and is all the more interesting from the present perspective, in that the percept is of a kind that might plausibly be supported by collicular mechanisms. Note also that even a methodological advance such as that recently reported by Persaud and colleagues (Persaud et al. 2007) will have to contend with this difficulty of knowing what an experimenter's question does, in fact, mean to the person to whom it is put, a problem familiar to anthropologists. In sum, then, the evidence from studies of blindsight so far does not show that visual awareness cannot exist in the absence of visual cortex, and the phenomenon accordingly does not eliminate the possibility that such awareness might be implemented at brainstem levels.

R3. Wakefulness, responsiveness, and consciousness

The concept of wakefulness and its neural mechanisms also caused some problems, specifically in comments by **Coenen**, **Morin**, and **Piccinini**. As often happens in the technical employment of terms taken from ordinary language, the technical usage "wakefulness" does not correspond to what we normally mean when we use that word in unselfconscious speech. When we say that someone is awake, we ordinarily mean to include command of the full range of faculties that tend to become available to us when waking up in the morning, that is, seeing, hearing, volition, and conscious functioning more generally. That is not, however, the way the term is used in physiology and neurology, and particularly not when the term "wakefulness" is employed in the context most germane to our topic, namely, in the definition and diagnosis of the vegetative state (Andrews 1999). Here the usage is more specific; an individual whose eyes open as part of a functioning sleep-wake cycle is said to be awake. In order to qualify as "vegetative" this state of wakefulness must exclude consciousness. Let us, for the sake of clarity, call this state of *unconscious wakefulness* "physiological wakefulness."

In diagnosing the vegetative state one must exclude the possibility that *in addition to* being awake in this sense the patient might be conscious. The neurological tests for environmental responsiveness are motivated by this necessity. They are employed as *proxies* for consciousness in individuals belonging to a species whose conscious status is unproblematic (except in the context of certain notorious thought experiments), and who exhibit behavioral signs normally associated with consciousness (namely, an eyes-open phase in a sleep-wake cycle), but who lack the capacity for self-report because of neurological damage. Such rough and ready proxies cannot, of course, deliver a reliable verdict regarding the presence or absence of consciousness. Indeed, when clinical populations diagnosed as vegetative by their routine use are subjected to more rigorous scrutiny, erroneous diagnosis is found to be a frequent occurrence (Andrews et al. 1996; Childs et al. 1993; Tresch et al. 1991). Moreover, in overall terms, the diagnostic error exhibits a consistent direction, such that patients who are in fact conscious are more often classified as vegetative than the reverse (a circumstance of some interest in relation to the issue of consciousness in children with hydranencephaly).

This, then, is the context for my use of "awake" and "wakefulness" and "responsiveness" in the target article. I carefully avoid letting the term "awake" stand for "conscious," but I always add terms such as "seeing, hearing" or other references to *experience* when referring to a conscious mode of function. My appeal to sensory responsiveness is predicated on the clinical context outlined earlier, and it has as its background the role that is played by children with hydranencephaly in my treatment. It figures in my summary of the capacities of decorticate mammals, as well, for whom massive anatomical, physiological, and behavioral homologies support the presumption of a conscious mode of normal, waking, brain function (Seth et al. 2005). This makes the application of criteria derived from human clinical experience a reasonable approach in their case as well, at least provisionally.

That, however, does not mean that responsiveness or purposive behavior as such, and without the constraining contexts just outlined, are relevant to the assessment of the presence of consciousness. Spinal reflexes, the various tropisms, and other forms of responsiveness exhibited by plants and unicellular animals, and even nonliving systems such as thermostat-controlled central heating, should be enough to dispose of that possibility. Responsiveness certainly does not entail consciousness, but in certain clinical circumstances the presence of sensory responsiveness can move a patient from one diagnostic category to another. With that clarification, I hope to have disambiguated the usage of "wakefulness," and, in addition, to have removed any puzzlement occasioned by my treatment of responsive and purposively moving medusas as nonconscious, while at the same time suggesting that responsive and purposively moving children with hydranencephaly are conscious (**Behrendt**). In ultimate terms, the distinction between conscious and nonconscious can never be made in behavioral terms, but hinges on the presence of a functioning neural mechanism of consciousness.

R4. Emotion

The topic of feelings and emotions was mentioned in numerous commentaries, and served as the main theme of three of them, namely those of **Izard, Panksepp,** and **Van Honk, Morgan, & Schutter (Van Honk et al.)** It also figured more indirectly in those of **Morsella & Bargh, Northoff,** and **Watt.** In ordinary language, feelings and emotions are something one experiences, that is, they are treated as inherently conscious phenomena. As such, they are of central concern to any theory of consciousness, and were featured in the present proposal as one of the three principal domains of its selection triangle. The commentaries add a multifaceted treatment of the topic far beyond its sketchy inclusion in my target article, and the complementarity and agreement between that of Izard, focused on human data, and those with a more comparative cast, is a welcome reminder of the conserved nature of the foundations of our psychological make-up.

Each feeling/emotion "feels differently" and makes us want to do different things (Sachs 1967; see also

Devor). This is what I mean by their role as "biases" in the economy of consciousness, so well captured in the commentary by **Izard.** His inclusion of "interest" among the emotions is well taken, and can be used to illustrate the point. It makes us want to explore. In this capacity, it is of central importance to telencephalic mechanisms of learning, memory, and problem solving, but it too has subcortical moorings. They include the hypothalamus (Swanson 2000), the midbrain dopaminergic system (see the interesting summary in the commentary by **Aboitiz et al.**; Bunzeck & Düzel 2006), the brainstem underpinnings of the navigation system (Sharp et al. 2001), and the mesopontine state control nuclei, whose important cholinergic component is highlighted in the commentary by **Collerton & Perry**.

The action-oriented content of feelings/emotions bears on the question raised by **Panksepp** regarding how we are to conceive of the first origins of conscious organization. My suggestion is that the emotional, sensory, and action aspects of consciousness were linked from the outset by providing the functional reason for a specifically conscious mode of organization. **Izard** points to the sensory occasions for emotional reactions, which, once aroused, exert their regulatory effects on behavior. The "innate releasing mechanisms" of ethology, often subcortically organized, supply a rich source of comparative evidence in this regard. **Morsella & Bargh** provide striking illustrations of how the action outcome, and the need to resolve potential conflicts between independent systems in order to achieve it, is intimately related to whether a certain process intrudes on consciousness or not. This rationale may even extend to the visceral nervous system mentioned by Panksepp, in that those aspects of it that engage consciousness would seem to be those that in one form or another require action on the body or the external world. Hunger and thirst, for example, inherently engage all three components of the selection triangle, but even a vague feeling like intestinal distress may serve to halt the further ingestion of food that may have been its cause (see Morsella & Bargh's commentary).

From these reflections on the topic of emotion addressed by these commentators, I turn to what amounts to a modality, which in a sense straddles the boundary between an emotional and a sensory system, namely, pain. In its often-accurate localizing capacity it serves a sensory function, whereas in its prepotent hedonic strength it epitomizes emotion. No other modality, save olfaction (which figures in commentaries by **Freeman** and **Morsella & Bargh**), comes even close to this inherent coupling between sensory and affective domains.

R5. Pain

Three commentaries address various aspects of the complex of theoretical, empirical, and clinical issues surrounding the perception of pain at perinatal, as well as adult stages of development (**Anand, Devor, Brusseau & Mashour**). It is gratifying to have this response from clinically oriented investigators, because no phenomenon casts the issues raised in my target article into sharper relief than the experience of pain. The reason is presumably the biological importance of the information it conveys, serving to alert an animal to a condition whose continuation would lead to tissue damage and ultimately to death. The nociceptive system is accordingly given high priority among the brain's signalling systems, a priority reflected not only in the multiple mechanisms devoted to it along the neuraxis, from spinal reflexes to cortical representation (Prescott et al. 1999), but in the hedonic strength with which it intrudes on consciousness. Overall, the pain system delivers the most powerful of the emotional-motivational "biases" governing the "needs" domain of the selection triangle I propose as the key to conscious function.

The coupling of motivational urgency (need), appropriate defensive measures (action), and swift localization of the offending source with regard to body surface and its surrounding space (target) is acute in the case of pain, and accordingly, can be expected to make an early appearance in the evolution of life forms, as well as in ontogeny (in good agreement with the evidence for prenatal pain sensitivity discussed in the commentaries). It must have helped shape the "optic brain" at the outset of vertebrate phylogeny, and today we find it prominently represented among the midbrain members of the proposed selection triangle: not only in the periaqueductal gray matter (Behbehani 1995), but in the intermediate to deep layers of the superior colliculus, as well (Bittencourt et al. 2005; McHaffie et al. 1989; Redgrave et al. 1996a; 1996b; Telford et al. 1996; Wang et al. 2000).

An upper brainstem implementation of a mechanism of primary consciousness, sketched in my target article, may thus help resolve some of the conceptual and empirical problems encumbering an exclusively corticocentric approach to the *experience* of pain so incisively presented and discussed in the commentaries. Parallels are also apparent with problems surrounding the definition and diagnosis of the vegetative state. Indeed, pain may be the Achilles' heel of this clinical entity. When, for the first time, a coma patient opens his or her eyes following a sharp cutaneous pinch, and thus clinically qualifies as having emerged into a vegetative state (assuming no additional sensory responsiveness), are those eyes opened by "an unconscious brainstem reflex"? Or does their opening signify the first fleeting emergence of the patient into consciousness, propelled into that state more readily by pain than other senses because its hedonic and arousing power exceeds that of other senses for basic biological reasons?

Such questions may be difficult to answer, but they deserve our attention, not only for reasons of basic science, but because they are fraught with consequences for medical ethics. Taken together, the three "pain commentaries" provide a many-faceted and rich treatment which brings both of these aspects of the topic into focus. They substantially add to and expand upon the perspective I have tried to articulate.

R6. Concerns about remnant cortex

The commentaries by **Coenen, Collerton & Perry, Freeman,** and **Watkins & Rees** express concerns regarding the possible role of remnant cortex in the capacities expressed by children with hydranencephaly. The target article is very clear about its presence in these children,

and gives a number of reasons why this factor is unlikely to provide an adequate account of their behavior. One of these is the contrast between their visual as compared to their auditory responsiveness, for which an account based on brainstem mechanisms provides a fit, whereas a cortical one does not. Here, I only wish to add that the thorough documentation and study of the capacities of these children has barely begun. My summary account is a preliminary one and in no way definitive, though it is my hope that it may provide a stimulus for the systematic kind of study that eventually will issue in a comprehensive account of their capacities, including details about what contribution, if any, spared cortex may make to those capacities. There are, moreover, children who live without any cortex at all, and some are born entirely without telencephalon (anencephaly). Their capacities too await systematic study, which will help determine the extent to which remnant cortex may play a role in hydranencephaly.

Freeman also asks about the extent of cortical removal in the studies of experimentally decorticated animals. In the studies by Whishaw and Kolb cited in my target article neocortex plus the partly allocortical cingulate gyrus was always removed. Even more extensive ablations do not necessarily alter outcomes in broad terms. Thus, the mating ability of decorticate male rats is not reduced by including the hippocampus in the removal (Whishaw & Kolb 1983), and even more radical damage, such as total removal of all telencephalic tissue, does not prevent a rat from performing and learning in an avoidance test situation (Huston & Tomaz 1986). This, of course, does not mean that decorticate animals do not have deficits (see, e.g., Whishaw et al. 1981), nor that different extents of lesions do not make a difference in outcomes. Rather, the bearing of these interventions on the topic I explore is that a basic level of differentiated and coherent behavioral competence survives even complete cortical removal.

The above six topics, then, cover my general response to over-arching concerns reflected in the commentaries. They raise numerous additional issues that deserve serious consideration. I cannot hope to cover them all in this reply, but I will attempt to deal with a set of further specific issues on an individual basis, in the hope of adding precision and perhaps removing some misapprehensions.

R7. Other specific issues

I am in perfect agreement with **Barceló & Knight**'s detailed demonstration of frontal top-down influence on the centrencephalic system. I would only add that such control is exercised "in cortical terms," that is, on the basis of cortical information, and that cortical information may not always be decisive for the global control of behavior. Let us assume that while a macaque is reaching for a manipulandum to deliver its verdict regarding a visual pattern discrimination, it suffers a sudden, sharp sting from an insect which has worked its way into the laboratory undetected. The macaque will withdraw its hand and launch defensive measures, some of which may be initiated prior to the completion of a prefrontal information path. My suggestion, far from novel (see Prescott et al. 1999, and references therein), is that some of these "early" effects engage mesodiencephalic structures served by effector paths of their own, and that frontal engagement belongs to the swift follow-up by which cortical mechanisms assess the significance of the event. The latter process would engage the entire circuitry outlined in Figure 1 of the commentary. **Barceló & Knight**'s commentary reminds us of how closely the sophisticated attainments of cortical mechanisms are tied in to the highly conserved mesodiencephalic machinery, in this case, specifically to the intermediate layers of the superior colliculus, which figure prominently in my proposal.

Issues already covered in my first three general topics repeatedly apply to **Behrendt**'s commentary. Some further matters follow here. Behrendt refers to thalamocortically dependent phenomenal contents of consciousness such as dreams and hallucinations and asks how they might relate to the brainstem systems I outline. Such contents are of course thalamocortical (see my response to **Doesburg & Ward**), but the superior colliculus is likely to be engaged under these circumstances no less than is the inferior colliculus. The superior colliculus does not remain passively open to sensory afference irrespective of stages of the sleep-wake cycle or levels of vigilance. Like the thalamocortical complex, it is yoked to sleep-wake cycles through projections from the mesopontine state control nuclei, and its unit responsiveness and intrinsic interactions are exquisitely sensitive to sleep-wake stages, as well as to levels of anesthesia, something which applies to its deeper layers in particular (for recent examples, see Brecht et al. 1999; 2001; Wang et al. 2000).

The possibility that mesodiencephalic mechanisms implement a first form of conscious function does not rob unconscious processes of places to hide. As the commentary by **Morsella & Bargh** makes clear, the cerebral cortex itself is one of these "places." The basal ganglia are another, and there is room left for them in the mesodiencephalon as well, because the structures I invoke occupy only part of that territory. Nor should the cerebellum be overlooked in this connection. The abstract diagram of my Figure 5 includes three different and bi-directional principal interfaces for unconscious activity, explicitly noted in the legend.

Behrendt misrepresents my position on the nature of the evidence for consciousness in children with hydranencephaly by joining a quote from my text to a context to which the quoted words clearly do not belong. As reference to the target article (end of 7th paragraph of sect. 5) will show, it is the "absences" of absence epilepsy in these children that I call "a weighty piece of evidence regarding their conscious status" and not their expressions of pleasure or excitement – a very different matter, indeed.

Finally, the ego-center that plays a crucial role in my scheme should not be identified with self experience, if the latter is taken in its reflective sense (see Note 1, and **Northoff**). I invoke, in this connection, a striking expression of Schopenhauer's. Reference to the page I cite will show that Schopenhauer there says exactly what I claim him to be saying. **Behrendt** is correct, however, in identifying my position regarding consciousness with that of philosophical idealism, though I prefer not to use the term on account of the history of

controversy, misunderstanding, and misuse with which it has been burdened.

My general comment on my neglect of the forebrain in the target article, and the comments on wakefulness, apply to **Coenen**'s commentary. Here, I only want to note that Meeren et al. (2005, cited in the target article) differ from Penfield and Jasper regarding the mechanism of absence epilepsy, principally by demonstrating that absence seizures can be *initiated* from cortical locations (something for which Penfield and Jasper had seen no evidence when stimulating the exposed cortex electrically, although other forms of seizure were thus induced; Penfield & Jasper 1954), and not with regard to the involvement of subcortical structures as such.

Besides recalling my usual caveats regarding my neglect of the forebrain, **Doesburg & Ward**'s commentary gives me the opportunity to address the issue of how one is to conceive of the relationship between a putative brainstem mechanism of primary consciousness and "thalamocortical consciousness" in *phenomenal* terms. I skirt this issue in my target article, which gives only the most rudimentary sketch of some of the circuitry that relates the two anatomically, but does not venture to suggest what might follow in phenomenal terms from that relatedness. Does any aspect of mesodiencephalic phenomenal content "show up" among the contents of adult human consciousness?

I assume, as a matter of course, that the contents of adult human consciousness are largely products of the thalamocortical complex. Considering our visual sensory consciousness alone, it is cast in the format of a panoramic three-dimensional world filled with shaped objects in complex mutual relations. The telencephalon is needed to stage such spectacles, whether one relies upon an avian "wulst" or a mammalian neocortex to gain entry to them (cf. **Edelman**). Imagine now using a clever projection system which allows one to superimpose on that scene elaborated by the forebrain an appropriately scaled midbrain rendition of the same scene. The phenomenal content of the latter, I have suggested, may resemble "mere loci of motion in an otherwise featureless noise field" (sect. 4.2, para. 10). As such, it would have nothing substantial to add to the cortical phenomenology. In fact, it might not even be detected in such a superposition, though it might be detectable by appropriate psychophysical procedures.

If for no other reason than this "dwarfing by contrast," the phenomenal content of the mesodiencephalic mechanism is unlikely to make much of a contribution to the phenomenal content of creatures equipped with a massive thalamocortical complex. Yet, as the relative size of this complex shrinks with diminishing encephalization index across mammals and beyond them into vertebrates without a neocortex, that contrast will diminish apace, with ever more of a *relative* contribution to phenomenal consciousness being made by the upper brainstem mechanism. This is my alternative to the assumption that function "migrates" from midbrain to forebrain in the course of phylogeny ("corticalization of function"), an issue discussed with reference to consciousness by Sewards and Sewards (2000). My alternative obviates a need to invoke any form of active suppression of the midbrain content of consciousness as encephalization progresses. Every function stays intact where evolution provided a neural mechanism for it; yet, as new, sophisticated mechanisms evolve, synthesizing ever more impressive "reality simulations," interest or focal awareness naturally dwells where the richest information exists. In our case, that is the forebrain, whereas for a lamprey, it is likely to be its multimodal tectum.

Given that I incline to discount a substantial contribution of midbrain content to the phenomenal content of intact, adult human conscious contents, how is one to construe the fact that at the same time I hold that the mesodiencephalic system is "integral to the constitution of the conscious state"? The issue deserves a fuller discussion than I can provide here (see also my response to **Watkins & Rees**), but in all brevity, the reason is that the tandem arrangement of zona incerta/superior colliculus is an integral part of the real-time logistics of the functional economy of the forebrain. By being tied in to the relevant higher-order nuclei of the thalamus through direct and prominent projections from both colliculus (excitatory) and zona incerta (inhibitory), this tandem arrangement is bound to affect the actual moment-to-moment composition of the contents of adult human consciousness. In light of the ubiquitous intrinsic inhibitory connectivity of the zona incerta, its role in this regard is likely to include swift and categorical decisions among rival contenders for awareness at a truly global level of gating (substrates for which are scarce in the thalamocortical complex itself, though this issue too deserves a more thorough discussion than I can provide here). In view of the midline-straddling commissural connectivity of the zona incerta stressed in the target article, this global gating should extend across the midline, a point of potential importance for our understanding of neglect syndromes ("extinction on double simultaneous stimulation"; see Bender 1952).

In sum, the circuitry is there, exerting powerful synaptic effects on the higher-order nuclei of the thalamus (Bartho et al. 2002). In view of this, it would seem that no account of the neural mechanisms responsible for the moment-to-moment composition of the contents of human consciousness can be complete without incorporating the zona incerta/superior colliculus tandem circuitry in its scheme. It intrudes directly on forebrain function, not by adding its own phenomenal contents to the forebrain's contents, but by supplying directly to the higher-order thalamus a running account of its own dual distillate of widespread convergent afference. Since the thalamic nuclei they address in push-pull fashion are those most directly tied to attention, neglect,[2] and consciousness, input from the tandem circuitry forms part of the balance of forces through which rival claims for awareness are settled, and, if my proposal has any merit, even helps settle them. I am suggesting, in other words, that the mesodiencephalic circuitry is an integral part of the function of selection in the thalamocortical complex, which **Doesburg & Ward** briefly discuss in their final paragraph. This would make the midbrain, and not the dorsal thalamus, the "base" of even the most elaborated mechanism of consciousness, in good agreement with my proposal that it is from that base that it originally expanded by the addition of ever more sophisticated circuitry from a rostral direction.

Gardner's commentary brings up a number of issues at the interface of robotics and decision theory relevant to the theoretical background of my perspective. In my

target article I use "optimize" in the sense of "achieve savings," rather than in any mathematically defined sense of optimality. The claim is that equipped with the selection triangle interface, an animal will achieve a more efficient deployment of its orienting behavior than would be possible in its absence. Its very purpose is efficient real-time management of the many trade-offs and compromises enforced by the multiple needs, actions, and targets that must find matches as opportunities present themselves over time in a lively and unpredictable world. With regard to the seminal contributions of Brooks it should be noted that his programmatic introduction of layered control architectures specifically excluded the kind of mechanism I propose. Among his guiding principles we find "little sensor fusion" and "no central models" (Brooks 1986, concisely summarized in Prescott et al. 1999). Massive sensor fusion is manifestly present in the superior colliculus of all vertebrates, and is incorporated directly into the "central model" of my proposed "analog reality simulator" (its analog nature setting it apart from every such model cast in the form of symbolic representation, it should be noted). The only way to introduce such a "central model" without prejudice to the considerable advantages offered by layered control is to place its nodal machinery at the highest level of the control architecture, and let its output contribute what is more akin to a bias than a command in the control of behavior, as in the present proposal. Hence, the emphasis I place on identifying what in fact constitutes the "highest level" of the vertebrate brain in control terms (rather than in cognitive ones). That, of course, does not mean that I "locate" adult human consciousness in the midbrain. What I do is to locate the "base" of the thalamocortically expanded human reality simulator in the midbrain (for which, see my response to **Doesburg & Ward**). Concerning the possibility of "*in silico* consciousness" see my concluding remarks.

Regarding **Gilissen**'s query about self-recognition, I refer to my first and third general responses, with further details in my response to **Doesburg & Ward**. Gilissen's fascinating account of the competences of jumping spiders reminds us that analogy (similar solutions to similar selection pressures) is the companion to homology (similarity on account of shared ancestry) in the evolution of life forms. This makes it unsafe to assume that a conscious mode of function will be found only in our own evolutionary vicinity. It also tells us that the familiar relations of the vertebrate brain plan will give us little guidance when we go outside our own phylum looking for fellow conscious creatures. If my suggestion that the savings offered by the selection triangle drives the evolution of consciousness has any merit, we would nevertheless not be groping entirely in the dark when embarking on such excursions. A neural interface between action selection, target selection, and motivation, and in command of orienting, would be the first thing to look for, with scrutiny of its format coming next. Gilissen's account of jumping spiders tempts me to go looking for such an interface in a species of *Portia*, and I thank him for putting me on its trail.

Glassmann raises and alludes to so many interesting issues that a lengthy essay would be needed to cover their full sweep. Let me therefore pick out a few items only. His observation of a transient dissociation between pitch and yaw components of orienting in cats with large cortical lesions is interesting from the point of view of the centrality assigned to an intermediate spherical coordinate system for orienting responses in my treatment. He asks whether intermodal and intramodal plasticity occurs in the mesodiencephalon itself, and the answer is indeed yes. For a striking demonstration of intermodal plasticity in the tectum, see Hyde and Knudsen (2001). The issue is relevant to the question of learning in children with hydranencephaly, alluded to in my target article. The important topic of working memory and its close relation to consciousness, finally, was given far too cursory a treatment in my target article. Glassmann helps remedy this shortcoming, with additional reference to the topic being provided by **Aboitiz et al.** and **Barceló & Knight.**

Krauzlis provides a concise summary of experimental evidence bearing on a collicular role in target selection, with special reference to its causal role. Particularly useful is his careful delimitation of what we know with some assurance from still unsettled issues. One of these is the extent to which the colliculus might reflect decisions made elsewhere rather than make them itself, an issue also raised in the commentaries by **Barceló & Knight** and **Prescott & Humphries.** There is no doubt about the prominence of its nigral and frontal cortical inputs in this regard; yet, considering the vast diversity of afferents converging on the colliculus below its stratum opticum, and the richness of interactions taking place within the colliculus itself, it would seem that these sources might not always be able to determine outcomes uniquely. However, as Krauzlis points out, that is an issue on which evidence is needed, promising another installment in the unfolding story of collicular competence so well summarized in his commentary.

I have repeatedly referred to the commentary by **Morsella & Bargh,** and here I want to add only a few comments. Their concern with identifying processes within the overall economy of brain function that enter consciousness and those that do not, and what may account for the difference, is a powerful tool in coming to grips with the nature and function of consciousness. I made some halting steps in this direction in a previous publication (Merker 2005), and find the examples provided by Morsella & Bargh to be both striking and apt. The approach could be extended into the compilation of a systematic inventory of such "included" and "excluded" functions. Their suggestion to exploit timing relations is well worth pursuing. It is an interesting fact in this connection that the direct retinal projection to the colliculus and the very indirect one via lateral geniculate, visual cortex, and thence to colliculus are roughly matched in their collicular "arrival time" (Berson 1988; Waleszczyk et al. 1999). In terms of the approach more generally, I would hesitate, however, to rely directly on the more extreme of the time estimates provided by Libet (see Libet et al. 1979), on account of the problems encumbering their interpretation (Pockett 2006, provides an entry to this issue).

My general comments on deliberately neglecting the telencephalon (commissures included) and comments on the term "wakefulness" both apply to **Morin**'s commentary, but some specific errors contained in it deserve additional notice. He mistakenly asserts that my definition of consciousness excludes its self-reflective form. In my use of the Indian "scale of sentience" for definitional

purposes, I say that "Each 'stage' in this scale, from mere experienced sensation to self-consciousness, falls within the compass of consciousness as here defined, and presupposes it" (target article, sect. 1, para. 5). Its fourth stage reads, "So this is I who am affected by this which is so" (sect. 1, para. 4), an admirably concise formulation of self-reflective awareness. I go on to refer to animals with advanced degrees of encephalization as the likely possessors of this form of awareness. In my definition I include all possible forms of consciousness "from mere experienced sensation to self-consciousness," making this a "broad" definition, whereas Morin prefers to exclude all but its self-reflective ("full-blown") varieties from serious consideration.

Self-reflective consciousness is certainly a worthy topic of study, and if my reference to it as "akin to a luxury" seems disparaging, I apologize. But to be conscious is not necessarily to be self-conscious even in the case of adult humans, as **Morin** himself has usefully pointed out in connection with its intermittency in everyday circumstances (Morin 2006, p. 366). Moreover, by neglecting the distinctions drawn in my general comment on wakefulness and responsiveness, Morin erroneously claims that neurophysiological evidence has supported the conclusion that consciousness is possible without a cortex for quite some time now. It is only for "physiological wakefulness" that such agreement exists, a state which is a presupposition for consciousness but does not include it (see my general comment on wakefulness). It therefore falls outside the compass of my definition of consciousness.

To avoid the danger of misidentifying aspects unique to a specialized form of consciousness as generic attributes of consciousness itself, a broad sampling of valid exemplars is desirable. Moreover, a focus on "full-blown" instances raises the question "whose full-blownness?" Are we to exclude patients with global aphasia from exhibiting any awareness worth our consideration because they lack "full-blown" human consciousness, which perforce includes language competence? Better, then, to first abstract a common denominator of conscious states from their many forms (Merker 1997), and let what they share rather than what divides them inform our conception of the nature of conscious function.

As far as I can tell, **Piccinini** has understood the main lines of my proposal. With reference to his fourth paragraph, I hope that it is clear that I think that the conscious contents of children with and without hydranencephaly differ, though both are of a phenomenal kind. I do not, however, think that the distinction between them relates to the philosophical distinction between creature consciousness and state consciousness. The reason is that I think McBride (1999) was correct in pointing out that the philosophical distinction is in the nature of a grammatical difference pertaining to how we use the word "consciousness" in different situations, and does not correspond to different psychological or ontological kinds of consciousness.

Prescott & Humphries' challenging commentary gives me an opportunity to clarify some essential aspects of the "selection triangle" sketched in my proposal, because they have misread its components. These are not hypothalamus, periaqueductal gray, and colliculus, nor does the zona incerta replace the basal ganglia as the principal mechanism for action selection in my scheme. The action selection vertex of the proposed selection triangle is explicitly assigned to the basal ganglia, as follows: "The third member of the selection triangle enters this system through the prominent projections from the substantia nigra to the intermediate collicular layers [refs.]. Here the final distillate of basal ganglia action-related information is interdigitated with the latticework of histochemically defined compartments that organize the input-output relations of the intermediate colliculus" (sect. 4.2 of the target article). That is, hypothalamus and periaqueductal gray are both part of the "motivation/emotion" vertex of the triangle, its action selection domain being occupied by the basal ganglia by way of the substantia nigra, with the superior colliculus itself supplying the target selection vertex.

I did not introduce the selection triangle as a mechanism for action selection, but as a mechanism of consciousness. As such, it takes the output of action selection as only one of its three principal inputs; and by interfacing the three within a unitary coordinate framework tying together ego-center, body, and world, it delivers a higher-order informational quantity (say in the form of a vector in a multidimensional space) that is added as a final optimizing bias to the global control of behavior, principally as a means of enhancing the *economy* of orienting behavior. That control – action selection included – could and perhaps would take place without it, but with a reduction (of unknown magnitude) in the efficiency of the overall deployment of orienting behavior towards the satisfaction of needs, according to my proposal.

A more precise specification of the "higher-order informational quantity" generated by the selection triangle awaits the formal modelling of the neural reality simulator, and ultimately its mathematical formalization. Presumably, dynamic interactions at the selection triangle interface are such as to define a unique location within it at each successive moment of psychological time, a location which, informally speaking, would indicate a direction of "prevailing concern or preoccupation of the moment." Such matters are, of course, not always reflected in overt behavior, which is one of the difficulties in dealing with consciousness rather than behavior. The closest that I can come to a generic characterization of its nature at this point would be "consequentiality," if one includes within the scope of that term both innate and acquired grounds for what might "matter" to an animal (for an acquired aspect, see Merker 2004a, pp. 572–73).

It is as a mechanism of selection among competing moment-to-moment bids for this hypothetical quantity that the zona incerta offers a highly suggestive connectivity, rather than for selection among actions themselves. The zona incerta adds a second external source of inhibition of collicular circuitry to that of the substantia nigra. The functional consequences of this dual external inhibition in the setting of the incompletely known complexities of intrinsic collicular circuitry are currently unknown and need to be elucidated before we can know who might dominate whom (and in what circumstances) in the intricacies of mesodiencephalic connective relations. For example, I do not think it is safe to assume that there are no conditions under which one or more of the numerous excitatory inputs converging upon the colliculus might not carry signal strengths jointly sufficient to penetrate even a combined nigral-incertal inhibitory

screen. It is even difficult to know whether the notion of a single hegemon is applicable to the complex dynamics animating this richly interconnected territory.

None of this, of course, casts any doubt on **Prescott & Humphries'** conclusion that "the BG [basal ganglia] are therefore ideally placed to provide the required funnel from distributed cortical processing to sequential brainstem operation." Distributed cortical processing is, however, only one of many sources of information along the neuraxis converging upon the mesodiencephalon, and according to the logic of layered control, the telencephalic level sometimes may have to resign itself to being overridden *without even being consulted* when signals regarding elemental necessities activate equally elemental brain stem (or even spinal) remedies, as illustrated by the account of the multiple levels of control governing defensive behavior offered by Prescott and colleagues (Prescott et al. 1999; see also my response to **Barceló & Knight**; in such cases the cortex is, of course, informed, but "after the fact," as it were; see Merker 2005, p. 98).

An astounding diversity of direct afferents from the entire length of the neuraxis converge on the deeper reaches of the superior colliculus, as well as on the zona incerta, both of which would seem to enjoy a truly global or "synoptic" view of CNS activity (see Edwards [1980] and references therein; Mitrofanis 2005). Let us remember, also, that the early vertebrate striatum lacked a globus pallidus and ventral tegmental area/substantia nigra output system proper. It relied instead on the nucleus tuberculi posterioris and the ventral thalamus of comparative terminology to translate its decisions into behavior (Grillner et al. 2005; Pombal et al. 1997; Smeets et al. 2000). The zona incerta of mammals is a direct derivative of this ventral thalamus, and supplies an, until recently, unsuspected source of powerful GABAergic inhibition to both thalamus and colliculus. Our understanding of functional relations among mesodiencephalic structures will remain incomplete until its contribution has been systematically charted.

Re-reading my account in the light of the commentary provided by **Prescott & Humphries,** I can see that in my target article I could have made a clearer distinction between the arguments by which I seek to establish the general point of the mesodiencephalon ("optic brain") as a nexus of superordinate control revolving around "integration for action" (not to be equated with action selection proper), on the one hand, and the additional – and conceptually distinct – discourse through which I introduce my selection triangle conception of the mechanism of consciousness, on the other. By partly assimilating the two I may have invited some of the misunderstandings I have tried to clear up here, and I thank the authors for giving me occasion to be more precise about the unique contribution I think a reality simulator cast in conscious format may make to the brain's functional economy.

The commentary by **Schlag** reminds us that mammals have (added?) collicular layers above the stratum opticum, which covers the surface of the colliculus in other vertebrates. The role of this superficial colliculus, and its relation to the deeper layers has been much debated in the past. In some ways it resembles a displaced thalamic nucleus intimately related to the posterior portion of the higher-order thalamic nuclei. But it is also connected to the deeper layers anatomically (see, e.g., Behan & Appell 1992), a connection which is functional (Doubell et al. 2003; Özen et al. 2000), and is unmasked by blocking inhibitory influences by bicuculline (Isa et al. 1998). As a direct contributor to the deeper layers, the superficial colliculus belongs to my scheme, though in agreement with Schlag's conclusion, the selection triangle interface of that scheme involves the deeper layers preferentially. I have considered the bearing of blindsight on consciousness theory in my general comments; and comments related to Schlag's suggestion regarding phylogeny in relation to hydranencephaly can be found in my response to **Doesburg & Ward.**

Seth gives a concise summary of one coherent proposal for why the unique connectivity of the cerebral cortex should be accorded a central place in the constitution of consciousness. As mentioned in my general introductory statement and in my response to **Doesburg & Ward,** I am in full agreement with assigning it such a role, as long as that role is not taken to exclude the possibility that a conscious mode of function may be implemented by other means. Naturally, when I state in the introduction of the target article that the functional utility of such an alternative implementation is independent of the sophistication with which its contents are elaborated, I mean only that it possesses functional utility, even in a rudimentary implementation, and not that increasing its sophistication would not enhance its utility.

That cortical connectivity possesses a distinctive "signature" with interesting characteristics has been revealed by measures of "mutual information" and other quantitative methods in the studies cited in **Seth's** commentary. But how are we to know that this signature provides a better fit with the characteristics of consciousness than with alternative functions, other than on intuitive grounds? We have no metric by which to assess the type of complexity possessed by consciousness, and in the absence of a quantitative method for determining "goodness of fit" between the two, alternatives might be worth considering. One such alternative is that the graph theoretic characteristics of cortical connectivity provide an optimal structure for information storage in memory, along lines I have presented in an earlier publication (Merker 2004a). In fact, the combination of differentiation with integration in cortical connectivity would seem to issue directly in that felicitous combination of item specificity with classificatory generality in memory storage, which I there propose as a unique advantage of specifically cortical connectivity. The issue seems worth exploring further.

The commentary by **Watkins & Rees** adds much valuable detail and a number of challenges. Of the latter, the ones based on blindsight may at least in part support a collicular role in awareness rather than challenge it, in light of the demonstration by Stoerig and Barth (2001) that GY is not phenomenally blind in his affected visual field (see my general comment on blindsight). That is, the collicular activity seen in connection with stimulus presentation in what was formerly referred to as GY's "blind" field may be the very neural activity that, in fact, constitutes his visual percept, though other possibilities are not at this time excluded. Since GY was also the subject for the experiment involving emotional faces cited by the commentators, the new blindsight results may affect its interpretation, as well (see commentary by **Izard**, and Reddy et al. 2006). Note, in this connection, that the

deeper layers of the superior colliculus receive afference from emotion-related brainstem circuitry (sect. 4.2 and Fig. 4 of the target article), an integral aspect of the selection triangle scheme.

Concerning the S-cone example, nothing I am aware of having stated implies a collicular role in which, say, direct afference from a peripheral source is a condition for its responsiveness to information supplied by that source, as if the collicular system worked in isolation prior to involvement in a change in conscious contents. On the contrary, much information reaches it indirectly, and it was above all its massive receipt of monosynaptic cortical afference, in layer upon layer throughout its depth, that I had in mind when suggesting that it supplies an essential step in the process by which one content of consciousness replaces another. It lies as an interposed filter in the path by which the descending output of layer 5 pyramidal cells returns to the cortex via the higher-order thalamic nuclei, after drastic compression in the mesodiencephalic bottleneck. It is in this position that I consider it to lie "in the loop" of a process that constitutes the contents of adult human consciousness, as discussed in my response to **Doesburg & Ward** and further on here. The direct path from cortical layer 5 to the superior colliculus is also a potential factor in conscious percepts evoked by direct electrical stimulation of the visual cortex, mentioned in the commentary.

The case reported by Weddell (2004) was cited in passing in my target article for its extension of evidence for the Sprague effect to humans. The rarity of comparable studies after neurological damage focused on the colliculi (itself rare, cf. commentary by **Schlag**) makes it important indeed, but its details are complicated. The patient exhibited a stage-wise sequence of neglect (left or right depending on stage) and other perceptual changes associated with the growth of a dorsal midbrain tumor in combination with frontocortical damage incurred during emergency shunting. In the course of its progression, the tumor invaded the thalamus, and the exact extent of collicular damage at different anatomical levels and stages of progression is unclear. Weddell's account of his findings is a tour-de-force of neurological inference, but it had to rely on numerous assumptions for which direct evidence is lacking. That said, the case provides evidence on the consequences of collicular damage, which in some respects supports the collicular role in adult human consciousness that I have suggested, and in other respects requires its revision.

It is noteworthy that the upper brainstem tumor damage exerted its primary effect on the type of forebrain functions tested in assessing neglect, which reflect competitive and selective processes in a number of ways (see, e.g., Geng & Behrmann 2006; Bender 1952). As I hope to have made clear, above all in my response to **Doesburg & Ward**, it is not by adding any conspicuous phenomenal content of its own to forebrain phenomenal consciousness that I regard the mesodiencephalic system to be integral to the constitution of even adult human consciousness, but in terms of affecting its moment-to-moment composition through just such competitive and selective processes. I refer to the special relation of the zona incerta/superior colliculus to the higher-order thalamic nuclei in this connection, and Weddell invokes the tecto-pulvinar and tecto-reticular systems in his account of the neglect findings in his patient, in agreement with what I propose.

Watkins & Rees are perfectly correct, however, in pointing out that the results of visual field perimetry in this patient run counter to my prediction that *"one conscious content will not be replaced by another without involvement of the mesodiencephalic system."* The detection of a stimulus in the perimeter involves the replacement of one conscious content by another, and, assuming that at least the colliculus was, in fact, completely disconnected from the thalamocortical complex in this patient, this replacement of conscious content would have taken place without its assistance. Since there were content replacements that did depend on collicular involvement (i.e., those constituting the patient's neglect syndrome), it seems that distinctions are necessary regarding which types of content replacement, in fact, are dependent upon the mesodiencephalic mechanism I outline. A hint in this regard is provided by the nature of perimetry, in which stimuli typically are presented singly, and thus do not require competitive selection processes – processes which lie at the heart of my conception of a selection triangle. Though further research is indicated, I am indebted to Watkins & Rees for alerting me to this need to refine my conception of the interaction between the mesodiencephalic and thalamocortical systems.

With that, I have come to the end of my comments on specific issues on an individual basis. As the attentive reader will have noticed, a considerable portion of these responses has been devoted to clarifying misapprehensions and correcting mistakes. No commentator should therefore feel slighted by not having been thus noticed. On the contrary, that is likely to indicate large areas of agreement between us. I have, however, benefited from every commentary, and thank each author for their contribution.

R8. Concluding remarks

The large number of commentaries alluding to my neglect of the forebrain illustrates the point made in my target article concerning the hold that a corticocentric perspective exercises over current thinking about consciousness. As can be seen from my reply to **Doesburg & Ward** (sect. 7, para. 8), such a perspective may even have merit in the case of adult human consciousness. My concern is that it fails to provide adequate guidance to the comparative study of consciousness, as well as to our understanding of the status of brain-damaged patients with regard to conscious function. Let me summarize, then, my position in a way that would not have been possible without the stimulation provided by the many and varied commentaries.

The corticocentric perspective can, in roughest outline, be rendered by a formula according to which the brain's mechanism of consciousness consists of "a brainstem-based system of wakefulness" (in the sense of physiological wakefulness) plus "a cortex-based system of consciousness." My sense is that this formula needs to be differentiated along two dimensions, one pertaining to systems-level organization and one to phylogeny. To begin with systems organization, I think that the brainstem contribution to consciousness is a dual one, and not single. On the one hand, it supplies an enabling function responsible for maintenance of the waking state in the sense of

physiological wakefulness as part of the sleep-wake cycle. This is the brainstem function I refer to in the target article as unproblematic and well-established, following the pioneering work of Moruzzi and Magoun (1949). In its modern incarnation, it consists of the mesopontine state control nuclei (adrenergic locus coeruleus, cholinergic pedunculopontine, and laterodorsal tegmental nuclei, and serotonergic dorsal raphé) plus the ascending reticular activating system (itself a complex entity, as underscored by **Watt**).

To this I would add a second brainstem function, which on its own, in the absence of cerebral cortex, may support a primary form of phenomenal consciousness. It is implemented, I suggest, in the structural complex I call the selection triangle, composed of periaqueductal gray, superior colliculus, and substantia nigra, surrounding the midbrain reticular formation. The deeper colliculus supplies a core interface between these three, in intimate interaction with the zona incerta. To this dual brainstem organization, the thalamocortical complex adds an ever more sophisticated expansion of phenomenal content over and above the upper brainstem primary mode, in accordance with the relative extent of encephalization in different vertebrates. This expansion culminates in the elaborate perceptual and cognitive contents of consciousness exhibited by highly encephalized mammals, a content which, in a few forms, includes self-consciousness (great apes and perhaps a few species of cetaceans), with humans uniquely adding language, as well.

Encephalization, then, introduces the second, phylogenetic, dimension of conceptual differentiation, in the form of the consequences that different degrees of encephalization entail for the nature of conscious contents. Some categorical differences in conscious contents, such as that between a capacity for self-consciousness and its absence, are matters intrinsic to the telencephalon, reflecting different degrees of elaboration of the thalamocortical complex, a matter that was no more than mentioned in passing in my target article. All vertebrates have a telencephalon, though of astoundingly different relative size. It follows that differences in conscious contents between different species will largely reflect differences in telencephalic organization between them. By comparison, the primary mode I suggest to be common to them all on account of highly conserved brainstem circuitry would exhibit less variation across species (compare, however, the colliculus/tectum of a tree shrew or an owl with that of a lamprey!). Regarding the relationship between the phenomenal content of the primary mode and that of the consciousness serving a highly encephalized mammal such as ourselves, see my response to **Doesburg & Ward** (sect. 7, para. 8).

At the end of this odyssey, I return to the metaphor introduced early in my target article in the form of the Indian scale of sentience. A conscious mode of function organized at the brainstem level would, I suggest, be sufficient to encompass its first two "stages," up to and including "this is so" (say, stimulus direction with respect to the animal, qualitatively different emotions and their degrees, etc.). In light of **Northoff**'s incisive analysis it might even extend into the third stage of the scale. The thalamocortical complex would cover an elaboration of its first three stages, and in some highly encephalized species, would add the fourth stage as well. At whatever stage of sophistication, these contents are coherently organized in nested fashion around an implicit ego-center supplying the origin of the nesting coordinate system – an arrangement whose format, I suggest, defines consciousness.

It is my hunch that any creature, or device for that matter, that would get about in the world as efficiently as a vertebrate without using more neural resources (or their silicon equivalent) than that vertebrate, would have to be equipped with the kind of analog reality simulator so far merely sketched in my account, but susceptible to further development in more formal terms. Thus equipped, it would be conscious, because its implicit ego-center would anchor a perspectival view coherently relating a simulated body to a simulated world. These latter entities, whether simply implemented or elaborate, and whether cast in a neural medium or eventually in silicon, are synthetic ones, contrived as efficiency measures in action control for the fulfillment of needs. It is the format, and not the medium of its implementation, that determines conscious status, I suggest.

I have learned much from my reading of the many and interesting commentaries, and from responding to them. It seems to me that by now the complete *BBS* treatment of my topic has arrived at a point where, at least, it will be difficult to misunderstand what I am in fact proposing. It gives me tremendous satisfaction to have had this opportunity for clarification, and I thank every commentary author for helping me to come to this point, and *Behavioral and Brain Sciences* for providing a forum for conducting exercises such as this.

Dedication: I dedicate my author's response to the memory of Heather Joy Krueger (18 January 2001 – 2 March 2007).

NOTES

1. This is not to be confused with self-consciousness (or reflective self-consciousness in **Northoff**'s more precise terminology, which represents a substantive advance over that employed in my target article).

2. For a recent contribution to the mechanisms of neglect relevant to the present perspective, not cited in the target article, see Rushmore et al. (2006).

References

[The letters "a" and "r" before author's initials stand for target article and response references, respectively.]

Abe, J. A. & Izard, C. E. (1999) The developmental functions of emotions: An analysis in terms of differential emotions theory. *Cognition and Emotion* 13:523–49. [CI]

Abeles, M. (1982) Role of the cortical neuron: Integrator or coincidence detector? *Israel Journal of Medical Science* 18:83–92. [aBM]

Aboitiz, F., López, V., López-Calderón, J. & Carrasco, X. (2006) Beyond Endophenotypes: An interdisciplinary approach to attentional deficit-hyperactivity disorder. In: *Focus in cognitive psychology research*, ed. M. A. Vanshevsky, pp. 110–123. Nova Science. [FA]

Adams, D. B. (1979) Brain mechanisms for offense, defense and submission. *Behavioral and Brain Sciences* 2:201–41. [aBM]

Adrian, E. D., Bremer F. & Jasper, H. H., eds. (1954) *Brain mechanisms and consciousness*. Charles C. Thomas. [aBM]

Albano, J. E. & Wurtz, R. H. (1978) Modification of the pattern of saccadic eye movements following ablation of the monkey superior colliculus. *Neuroscience Abstracts* 4:161. [aBM]

Albers, F. J. & Meek, J. (1991) Dendritic and synaptic properties of collicular neurons: A quantitative light and electron microscopical study of Golgi-impregnated cells. *Anatomical Record* 231:524–37. [aBM]

Allport, D. A. (1987) Selection for action: Some behavioral and neurophysiological considerations of attention and action. In: *Perspectives on perception and action*, ed. H. Heuer & A. F. Sanders, pp. 395–419. Erlbaum. [aBM]

Altman, J. S. & Kien, J. (1989) New models for motor control. *Neural Computation* 1:173–83. [aBM]

An, X., Bandler, R., Ongür, D. & Price, J. L. (1998) Prefrontal cortical projections to longitudinal columns in the midbrain periaqueductal gray in macaque monkeys. *Journal of Comparative Neurology* 401:455–79. [aBM]

Anand, K. J. S. & Aynsley-Green, A. (1985) Metabolic and endocrine effects of surgical ligation of patent ductus arteriosus in the human preterm neonate: Are there implications for further improvement of postoperative outcome? *Modern Problems in Paediatrics* 23:143–57. [KJSA]

Anand, K. J. S. & Carr, D. B. (1989) The neuroanatomy, neurophysiology, and neurochemistry of pain, stress, and analgesia in newborns and children. *Pediatric Clinics of North America* 36(4):795–822. [KJSA]

Anand, K. J. S. & Craig, K. D. (1996) New perspectives on the definition of pain. *Pain* 67(1):3–6; discussion 209–11. [KJSA]

Anand, K. J. S. & Hickey, P. H. (1987) Pain and its effects in the human neonate and fetus. *New England Journal of Medicine* 317:1321–29. [aBM]

Anand, K. J. S., Rovnaghi, C., Walden, M. & Churchill, J. (1998) Consciousness, behavior, and clinical impact of the definition of pain. *Pain Forum* 8(2): 64–73. [KJSA]

Anderson, J. R. (2001) Self and others in nonhuman primates: A question of perspective. *Psychologia* 44:3–16. [EG]

Andre, M., Flenat, F., Floquet, J. & Picard, L. (1975) Major cerebral lesions with normal neonatal neurologic behavior. *Archives Francaises de Pediatrie* 32:915–24. (In French.) [aBM]

Andrews, K. (1990) The vegetative state – clinical diagnosis. *Postgraduate Medical Journal* 75:321–24. [rBM]

Andrews, K., Murphy, L., Munday, R. & Littlewood, C. (1996) Misdiagnosis of the vegetative state: Retrospective study in a rehabilitation unit. *British Medical Journal* 313:13–16. [rBM]

Ashton, H. (2002) Motivation: Reward and punishment systems. In: *The neurochemistry of consciousness*, ed. E. Perry, H. Ashton & A. Young, John Benjamins. [JvH]

Baars, B. J. (1988) *A cognitive theory of consciousness*. Cambridge University Press. [aBM, AM, JvH]

(1993) How does a serial, integrated and very limited stream of consciousness emerge from a nervous system that is mostly unconscious, distributed, parallel and of enormous capacity? In: *Theoretical and experimental studies of consciousness, Ciba Foundation Symposium 174*, ed. G. Bock & J. Marsh, pp. 282–90. Wiley. [aBM]

(2002) The conscious access hypothesis: Origins and recent evidence. *Trends in Cognitive Sciences* 6:47–52. [EM]

Baars, B. J., Ramsoy, T. Z. & Laureys, S. (2003) Brain, conscious experience and the observing self. *Trends in Neurosciences* 26:671–75. [aBM]

Baddeley, A. D. (1992) Consciousness and working memory. *Consciousness and Cognition* 1:3–6. [aBM]

Baerends, G. P. (1976) The functional organization of behaviour. *Animal Behaviour* 24:726–38. [aBM]

Bagchi, A. (1975) *Indian definition of mind*. Sanskrit College Research Series, No. XCVIII. Sanskrit College. [aBM]

Ballard, C. G., Court, J. A., Piggott, M., Johnson, M., O'Brien, J., McKeith, I., Holmes, C., Lantos, P., Jaros, E., Perry, R. & Perry, E. (2002) Disturbances of consciousness in Dementia with Lewy Bodies associated with alteration in nicotinic receptor binding in the temporal cortex. *Consciousness and Cognition* 11:461–74. [DC]

Bandler, R. & Keay, K. A. (1996) Columnar organization in the midbrain periaqueductal gray and the integration of emotional expression. *Progress in Brain Research* 107:285–300. [aBM]

Bandyopadhyay, S., Gonzalez-Islas, C. & Hablitz, J. J. (2005) Dopamine enhances spatiotemporal spread of activity in rat prefrontal cortex. *Journal of Neurophysiology* 93:864–72. [TA]

Barceló, F. & Knight, R. T. (2000) Dorsolateral prefrontal lesions alter the predictability and distractibility value of novel stimuli in a visual target detection task. *Society of Neuroscience Abstracts* 2: 839.11. [FB]

(in press) An information theoretic approach to context coding and entropy control in the human prefrontal cortex. *Cerebral Cortex*. [FB]

Barceló, F., Escera, C., Corral, M. J. & Perianez, J. A. (2006) Task switching and novelty processing activate a common neural network for cognitive control. *Journal of Cognitive Neuroscience* 18:1734–48. [FB]

Barceló, F., Periañez, J. A. & Knight, R. T. (2002) Think differently: A brain orienting response to task novelty. *NeuroReport* 13:1887–92. [FB]

Barceló, F., Suwazono, S. & Knight, R. T. (2000) Prefrontal modulation of visual processing in humans. *Nature Neuroscience* 3:399–403. [FB]

Bard, P. (1928) A diencephalic mechanism for the expression of rage with special reference to the sympathetic nervous system. *American Journal of Physiology* 84:490–515. [aBM]

Bard, P. & Rioch, D. M. (1937) A study of four cats deprived of neocortex and additional portions of the forebrain. *Bulletin of the Johns Hopkins Hospital* 60:73–148. [WJF, aBM]

Barsalou, L. W. (2003). Situated simulation in the human conceptual system. *Language and Cognitive Processes* 18:513–62. [EM]

Bartocci, M., Bergqvist, L. L., Lagercrantz, H. & Anand, K. J. S. (2006) Pain activates cortical areas in the preterm newborn brain. *Pain* 122(1-2): 109–17. [KJSA]

Bartho, P., Freund, T. F. & Acsady, L. (2002) Selective GABAergic innervation of thalamic nuclei from zona incerta. *European Journal of Neuroscience* 16:999–1014. [arBM]

Bassett, J. P. & Taube, J. S. (2001) Neural correlates for angular head velocity in the rat dorsal tegmental nucleus. *Journal of Neuroscience* 21:5740–51. [aBM]

Basso, M. A., Powers, A. S. & Evinger, C. (1996) An explanation for reflex blink hyperexcitability in Parkinson's Disease. 1. Superior colliculus. *The Journal of Neuroscience* 16:7308–17. [DC]

Basso, M. A. & Wurtz, R. H. (1997) Modulation of neuronal activity by target uncertainty. *Nature* 389:66–69. [RJK]

(1998) Modulation of neuronal activity in superior colliculus by changes in target probability. *Journal of Neuroscience* 18:7519–34. [aBM]

(2002) Neuronal activity in substantia nigra pars reticulata during target selection. *Journal of Neuroscience* 22:1883–94. [aBM]

Bastian, J. (1982) Vision and electroreception: Integration of sensory information in the optic tectum of weakly electric fish *Apteronotus albifrons*. *Journal of Comparative Physiology* 147:287–98. [aBM]

Bauer, P. J. (2006) Constructing a past in infancy: A neuro-developmental account. *Trends in Cognitive Sciences* 10:175–81. [CI]

Beach, F. A., Hebb, D. O., Morgan, C. T. & Nissen, H. W., eds. (1960) *The neuropsychology of Lashley. Selected papers of K. S. Lashley*. McGraw-Hill. [RBC]

Behan, M. & Appell, P. P. (1992) Intrinsic circuitry in the cat superior colliculus: Projections from the superficial layers. *Journal of Comparative Neurology* 315:230–43. [rBM]

Behan, M. & Kime, N. M. (1996) Intrinsic circuitry in the deep layers of the cat superior colliculus. *Visual Neuroscience* 13:1031–42. [aBM]

Behbehani, M. M. (1995) Functional characteristics of the midbrain periaqueductal gray. *Progress in Neurobiology* 46:575–605. [arBM]

Behrendt, R. P. (2003) Hallucinations: synchronisation of thalamocortical gamma oscillations underconstrained by sensory input. *Consciousness and Cognition* 12:413–51. [R-PB]

(2004) A neuroanatomical model of passivity phenomena. *Consciousness and Cognition* 13:579–609. [R-PB]

(2005) Passivity phenomena: Implications for the concept of self. *Neuro-Psychoanalysis* 7:185–207. [R-PB]

(2006) The subjectivity of the perceived world: Psychopathology and the mind-body problem from a perspective of idealism. In: *New developments in consciousness research*, ed. Vincent W. Vallio, pp. 189–209. Nova Science. [R-PB]

Beitz, A. J. (1982) The organization of afferent projections to the midbrain periaqueductal gray of the rat. *Neuroscience* 7:133–59. [aBM]

Bell, A. H., Corneil, B. D., Munoz, D. P. & Meredith, M. A. (2003) Engagement of visual fixation suppresses sensory responsiveness and multisensory integration in the primate superior colliculus. *European Journal of Neuroscience* 18: 2867–73. [aBM]

Bell, M. L. (1997) Postoperative pain management for the cognitively impaired older adult. *Seminars in Perioperative Nursing* 6(1):37–41. [KJSA]

Benatar, D. & Benatar, M. (2001) A pain in the fetus: Toward ending confusion about fetal pain. *Bioethics* 15(1):57–76. [KJSA]

Bender, M. B. (1952) *Disorders in perception*. Charles C. Thomas. [rBM]

Benevento, L. A. & Fallon, J. H. (1975) The ascending projections of the superior colliculus in the rhesus monkey (*Macaca mulatta*). *Journal of Comparative Neurology* 160:339–62. [aBM]

Benson, D. L., Isackson, P. J., Gall, C. M. & Jones, E. G. (1992) Contrasting patterns in the localization of glutamic decarboxylase and Ca^{2+}/calmodulin protein kinase gene expression in the rat central nervous system. *Neuroscience* 46:825–49. [aBM]

Benson, D. L., Isackson, P. J., Hendry, S. H. & Jones, E. G. (1991) Differential gene expression for glutamic acid decarboxylase and type II calcium-calmodulin-dependent protein kinase in basal ganglia, thalamus, and hypothalamus of the monkey. *Journal of Neuroscience* 11:1540–61. [aBM]

Berson, D. M. (1988) Retinal and cortical inputs to cat superior colliculus: Composition, convergence and laminar specificity. *Progress in Brain Research* 75:17–26. [rBM]

Berson, D. M. & McIlwain, J. T. (1983) Visual cortical input to deep layers of cat's superior colliculus. *Journal of Neurophysiology* 50:1143–55. [aBM]

Bezard, E., Dovero, S., Prunier, C., Ravenscroft, P., Chalon, S., Guilloteau D., Crossman, A. R., Bioulac, B., Brotchie, J. M. & Gross, C. F. (2001) Relationship between the appearance of symptoms and the level of nigrostriatal

degeneration in a progressive 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine-lesioned macaque model of Parkinson's disease. *Journal of Neuroscience* 21:6853–61. [BSC]

Binns, K. E. (1999) The synaptic pharmacology underlying sensory processing in the superior colliculus. *Progress in Neurobiology* 59:129–59. [aBM]

Bisti, S., Maffei, L. & Piccolino, M. (1972) Variations of the visual responses of the superior colliculus in relation to body roll. *Science* 175:456–57. [aBM]

Bittencourt, A. S., Nakamura-Palacios, E. M., Mauad, H., Tufik, S. & Schenberg, L. C. (2005) Organization of electrically and chemically evoked defensive behaviors within the deeper collicular layers as compared to the peri-aqueductal gray matter of the rat. *Neuroscience* 133:873–92. [arBM]

Bjursten, L.-M., Norrsell, K. & Norrsell, U. (1976) Behavioral repertory of cats without cerebral cortex from infancy. *Experimental Brain Research* 25: 115–30. [aBM]

Block, N. (1995) On a confusion about a function of consciousness. *Behavioral and Brain Sciences* 18:227–87. [AMLC, JvH]

(1996) How can we find the neural correlate of consciousness? *Trends in Neuroscience* 19(11):456–59. [GN]

(2005) Two neural correlates of consciousness. *Trends in Cognitive Sciences* 9:46–52. [CI]

Blumberg, B. (1994) Action-selection in Hamsterdam: Lessons from ethology. In: *The Third International Conference on the Simulation of Adaptative Behavior*, ed. D. Cliff, P. Husbands, J. A. Meyer & S. W. Wilson, pp. 108–17. MIT Press. [aBM]

Blumenfeld, H. & Taylor, J. (2003) Why do seizures cause loss of consciousness? *The Neuroscientist* 9:301–10. [aBM]

Boeve, B. F., Silber, M. H. & Ferman, T. J. (2004) REM sleep behavior disorder in Parkinson's Disease and Dementia with Lewy Bodies. *Journal of Geriatric Psychiatry and Neurology* 17:146–57. [DC]

Bogen, J. E. (1995) On the neurophysiology of consciousness. Part 1: Overview. *Consciousness and Cognition* 4:52–62. [aBM]

Boivie, J., Leijon, C. & Johansson, I. (1989) Central post-stroke pain: A study of the mechanisms through analysis of the sensory abnormalities. *Pain* 37: 173–85. [MD]

Bokor, H., Frere, S. G., Eyre, M. D., Slezia, A., Ulbert, I., Luthi, A. & Acsady, L. (2005) Selective GABAergic control of higher-order thalamic relays. *Neuron* 45:929–40. [aBM]

Bradshaw, J., Saling, M., Hopwood, M., Anderson, V. & Brodtmann, A. (2004). Fluctuating cognition in dementia with Lewy bodies and Alzheimer's disease is qualitatively distinct *Journal of Neurology, Neurosurgery and Psychiatry* 75:382–87. [DC]

Braitenberg, V. & Schüz, A. (1998) *Cortex: Statistics and geometry of neuronal connectivity*, 2nd edition. Springer-Verlag. [WJF]

Brandão, M. L., Anseloni, V. Z., Pandóssio, J. E. De Araújo, J. E. & Castilho, V. M. (1999) Neurochemical mechanisms of the defensive behavior in the dorsal midbrain. *Neuroscience and Biobehavioral Reviews* 23:863–75. [aBM]

Brann, C. B. (1966) The sensory biology of the living jawless fishes: A phylogenetic assessment. *Brain, Behavior and Evolution* 48:262–76. [aBM]

Breau, L. M., Camfield, C. S., McGrath, P. J. & Finley, G. A. (2004) Risk factors for pain in children with severe cognitive impairments. *Developmental Medicine and Child Neurology* 46(6):364–71. [KJSA]

Brecht, M., Singer, W. & Engel, A. K. (1998) Correlation analysis of cortico-tectal interactions in the cat visual system. *Journal of Neurophysiology* 79: 2394–407. [aBM]

(1999) Patterns of synchronization in the superior colliculus of anesthetized cats. *The Journal of Neuroscience* 19:3567–79. [arBM]

Brecht, M., Goebel, R., Singer, W. & Engel, A. K. (2001) Synchronization of visual responses in the superior colliculus of awake cats. *NeuroReport* 12:43–47. [arBM]

Brodmann, K. (1909) *Vergleichende lokalisationslehre der grosshirnrinde*. Barth. [WJF]

Brooks, J. C., Zambreanu, L., Godinez, A., Craig, A. D. & Tracey, I. (2005) Somatotopic organisation of the human insula to painful heat studied with high resolution functional imaging. *NeuroImage* 27(1):201–209. [KJSA]

Brooks, R. A. (1986) A robust layered control system for a mobile robot. *IEEE Journal on Robotics and Automation* 2:14–23. [RAG, arBM]

(1989) A robot that walks: Emergent behaviors from a carefully evolved network. *Neural Computation* 1:253–62. [RAG, aBM]

(1990) Elephants don't play chess. *Robotics and Autonomous Systems* 6: 3–15. [RAG]

(1991) Intelligence without representation. *Artificial Intelligence* 47: 139–59. [RAG]

(1994) Coherent behavior from many adaptive processes. In: *From animals to animats 3: Proceedings of the Third International Conference on Simulation of Adaptive Behavior*, ed. D. Cliff, P. Husbands, J.-A. Meyer & S. W. Wilson, pp. 22–29. MIT Press. [aBM]

(2002) *Flesh and machines: How robots will change us*. Vintage. [RAG]

Brown, J. W. (1988) *The life of the mind*. Erlbaum. [SMD]

(1989) Essay on perception. In: *Neuropsychology of perception*, ed. J. W. Brown, pp. 233–56. Erlbaum. [aBM]

Brudzynski, S. M., Cruikshank, J. W. & McEachlan, B. S. (1995) Cholinergic mechanisms in generalised seizures: importance of the zona incerta. *Canadian Journal of Neurological Sciences* 22:116–20. [aBM]

Bruner, J. S., Goodnow, J. J. & Austin, G. A. (1956) *A study of thinking*. Wiley. [CI]

Buck, L. B. (2000). Smell and taste: The chemical senses. In: *Principles of neural science*, 4th edition. ed. E. R. Kandel, J. H. Schwartz & T. M. Jessell. pp. 625–47. McGraw-Hill. [EM]

Bunzeck, N. & Düzel, E. (2006) Absolute coding of stimulus novelty in the human substantia nigra/VTA. *Neuron* 51:369–79. [rBM]

Bures, J., Buresov, O. & Krivnck, J. (1974) *The mechanism and applications of Leão's spreading depression of electroencephalographic activity*. Academic Press. [WJF]

Burnett, L. R., Stein, B. E., Chaponis, D. & Wallace, M. T. (2004) Superior colliculus lesions preferentially disrupt multisensory orientation. *Neuroscience* 124:535–47. [DC]

Butler, A. B. (2000) Sensory system evolution at the origin of craniates. *Philosophical Transactions of the Royal Society (London B)* 355:1309–13. [aBM]

Butler, A. B. & Cotterill, R. M. J. (2006) Mammalian and avian neuroanatomy and the question of consciousness in birds. *Biological Bulletin* 211: 106–27. [DBE]

Butler, A. B. & Hodos, W. (1996) *Comparative vertebrate neuroanatomy: Evolution and adaptation*. Wiley-Liss. [aBM]

Cabanac, M. (1992) Pleasure: The common currency. *Journal of Theoretical Biology* 155:173–200. [aBM]

(1996) The place of behavior in physiology. In: *Handbook of physiology, environmental physiology*, ed. M. J. Fregly & C. Blatteis, pp. 1523–36. Oxford University Press. [aBM]

Caduxsean, J. & Roger, M. (1985) Afferent projections to the superior colliculus in the rat, with special attention to the deep layers. *Journal für Hirnforschung* 26:667–81. [aBM]

Campbell, D. T. (1956) Perception as substitute trial and error. *Psychological Review* 63:330–42. [RBG]

(1966) Pattern matching as an essential in distal knowing. In: *The psychology of Egon Brunswick*, ed. K. R. Hammond, pp. 81–106. Holt, Rinehart & Winston. [RBG]

(1974) Evolutionary epistemology. In: *The philosophy of Karl Popper, vol. 14, I & II: The library of living philosophers*, ed. P. A. Schilpp, pp. 413–63. Open Court. [RBG]

Carello, C. D. & Krauzlis, R. J. (2004) Manipulating intent: Evidence for a causal role of the superior colliculus in target selection. *Neuron* 43(4):575–83. [RJK, aBM]

Carpenter, R. H. S. (1991) The visual origins of ocular motility. In: *Vision and visual dysfunction. Vol 8: Eye movements*, ed. R. H. S. Carpenter, pp. 1–10. MacMillan. [aBM]

Carrive, P., Bandler, R. & Dampney, R. A. (1989) Somatic and autonomic integration in the midbrain of the unanesthetized decerebrate cat: A distinctive pattern evoked by excitation of neurones in the subtentorial portion of the midbrain periaqueductal grey. *Brain Research* 483: 251–58. [aBM]

Carroll, S. B., Grenier, J. K. & Weatherbee, S. D. (2001) *From DNA to diversity: Molecular genetics and the evolution of animal design*. Blackwell Science. [DBE]

Carter, C. S., Witt, D. M., Kolb, B. & Whishaw, I. Q. (1982) Neonatal decortication and adult female sexual behavior. *Physiology and Behavior* 29:763–66. [aBM]

Casagrande, V. A. & Diamond, I. T. (1974) Ablation study of the superior colliculus in the tree shrew (*Tupaia glis*). *Journal of Comparative Neurology* 156: 207–38. [aBM]

Cavanaugh, J. & Wurtz, R. H. (2004) Subcortical modulation of attention counters change blindness. *Journal of Neuroscience* 24(50):11236–243. [RJK, aBM]

Chalmers, D. J. (1996) *The conscious mind: In search of a fundamental theory*. Oxford University Press. [CI, GN]

Charrand, V. L., Maddux, W. W. & Lakin, J. L. (2005) Beyond the perception-behavior link: The ubiquitous utility and motivational moderators of nonconscious mimicry. In: *The new unconscious*, ed. R. R. Hassin, J. S. Uleman & J. A. Bargh, pp. 334–61. Oxford University Press. [CI]

Childs, N. L., Mercer, W. N. & Childs, H. W. (1993) Accuracy of diagnosis of persistent vegetative state. *Neurology* 43:1465–67. [rBM]

Clancy, R. (1998) Electroencephalography in the premature and full-term infant. In: *Fetal and neonatal physiology*, ed. R. A. Polin & W. W. Fox, pp. 2147–65. W. B. Saunders. [RRB]

Closs, S. J., Barr, B. & Briggs, M. (2004) Cognitive status and analgesic provision in nursing home residents. *British Journal of General Practice* 54(509): 919–21. [KJSA]

Clower, D. M., West, R. A., Lynch, J. C. & Strick, P. L. (2001) The inferior parietal lobule is the target of output from the superior colliculus, hippocampus, and cerebellum. *Journal of Neuroscience* 21:6283–91. [aBM]

Coates, M. M. (2003) Visual ecology and functional morphology of Cubozoa (Cnidaria). *Integrative and Comparative Biology* 43:542–48. [SMD]

Coenen, A. M. L. (1995) Neuronal activities underlying the electroencephalogram and evoked potentials of sleeping and waking: Implications for information processing. *Neuroscience and Biobehavioral Reviews* 19:447–63. [AMLC]

(1998) Neuronal phenomena associated with vigilance and consciousness: From cellular mechanisms to electroencephalographic patterns. *Consciousness and Cognition* 7:42–53. [AMLC]

(1999) Similarities between deep slow wave sleep and absence epilepsy. *Sleep-Wake Research in The Netherlands* 10:99–103. [AMLC]

Coizet, V., Comoli, E., Westby, G. W. & Redgrave, P. (2003) Phasic activation of substantia nigra and the ventral tegmental area by chemical stimulation of the superior colliculus: An electrophysiological investigation in the rat. *European Journal of Neuroscience* 17:28–40. [FA]

Collerton, D., Perry, E. & McKeith, I. (2005) Why people see things that are not there: A novel Perception and Attention Deficit model for recurrent complex visual hallucinations. *Behavioral and Brain Sciences* 28(6):737–94. [DC]

Collins, C. E., Lyon, D. C. & Kaas, J. H. (2005) Distribution across cortical areas of neurons projecting to the superior colliculus in New World monkeys. *Anatomical Record* (Part A) 285A:619–27. [aBM]

Comoli, E., Coizet, V., Boyes, J., Bolam, J. P., Canteras, N. S., Quirk, R. H., Overton, P. G. & Redgrave, P. (2003) A direct projection from superior colliculus to substantia nigra for detecting salient visual events. *Nature Neuroscience* 6:974–80. [FA]

Corbetta, M. (1998) Frontoparietal cortical networks for directing attention and the eye to visual locations: Identical, independent, or overlapping neural systems? *Proceedings of the National Academy of Sciences USA* 95:831–38. [aBM]

Counter, S. A. (2005) Music stirred her damaged brain. *The Boston Globe, Health/Science*, March 29. 2005. Available at: http://www.boston.com/news/globe/health_science/articles/2005/03/29/music_stirred_her_damaged_brain [aBM]

Courjon, J.-H., Olivier, E. & Pélisson, D. (2004) Direct evidence for the contribution of the superior colliculus in the control of visually guided reaching movements in the cat. *Journal of Physiology* 556:675–81. [aBM]

Covington, C., Taylor, H., Gill, C., Padaliya, B., Newman, W., Smart, J. R. 3rd & Charles, P. D. (2005) Prolonged survival in hydranencephaly: A case report. *Tennessee Medicine* 98:423–24. [aBM]

Cowan, N. (2001) The magical number 4 in short-term memory: A reconsideration of mental storage capacity. *Behavioral and Brain Sciences* 24:87–185. [aBM]

Cowey, A. (2004) The 30th Sir Frederick Bartlett lecture: Fact, artefact, and myth about blindsight. *Quarterly Journal of Experimental Psychology* 57A: 577–609. [rBM]

Cox, P. H. (1996) *An initial investigation of the auditory egocenter: Evidence for a "cyclopean ear."* Doctoral dissertation. North Carolina State University, Raleigh, NC. [aBM]

Craig, A. D. (2003) Interoception: The sense of the physiological condition of the body. *Current Opinion in Neurobiology* 13(4):500–505. [KJSA]

Craig, K. D. (1997) Implications of concepts of consciousness for understanding pain behaviour and the definition of pain. *Pain Research and Management* 2(2):111–17. [KJSA]

Craik, F., Moroz, T., Moscovitch, M., Stuss, D., Winocur, G., Tulving, E. & Kapur, S. (1999) In search of the self: A positron emission tomography study. *Psychological Science* 10:26–34. [AM]

Crick, F. & Koch, C. (1998) Consciousness and neuroscience. *Cerebral Cortex* 8:97–107. [JvH]

(2003) A framework for consciousness. *Nature Neuroscience* 6:119–26. [aBM, JvH]

Crutcher, M. D., Branch, M. H. DeLong, M. R. & Georgopoulos, A. P. (1980) Activity of zona incerta neurons in the behaving primate. *Society for Neuroscience Abstracts* 6:676. [aBM]

Cuevas, K., Rovee-Collier, C. & Learmonth, A. E. (2006) Infants form associations between memory representations of stimuli that are absent. *Psychological Science* 17:543–49. [CI]

Cunningham, N. (1998) Comments on Derbyshire, PAIN, 67 (1996) 210–211 [letter comment]. *Pain* 74(1):102–106. [KJSA]

(1999) Inclusion of the nonverbal patient: A matter of moral emergency. *Pain Forum* 8:110–12. [KJSA]

Damasio, A. R. (1994) *Descartes' error*. G. P. Putnam. [JP, JvH]

(1999) *Feeling of what happens: Body, emotion, and the making of consciousness*. Harcourt Brace. [JvH, DEW]

Danober, L., Deransart, C., Depaulis, A., Vergnes, M. & Marescaux, C. (1998) Pathophysiologic mechanisms of genetic absence epilepsy in the rat. *Progress in Neurobiology* 55:27–57. [aBM]

Davis, M. & Whalen, P. J. (2001) The amygdala: Vigilance and emotion. *Molecular Psychiatry* 6:13–43. [JvH]

Dean, P., Porrill, J. & Stone, J. V. (2002) Decorrelation control by the cerebellum achieves oculomotor plant compensation in simulated vestibulo-ocular reflex. *Proceedings of the Royal Society of London, Series B* 269:1895–904. [aBM]

(2004) Visual awareness and the cerebellum: Possible role of decorrelation control. In: *The roots of visual awareness*, ed. C. A. Heywood, A. D. Milner & C. Blakemore. *Progress in Brain Research* 144:61–75. [aBM]

Dean, P. & Redgrave, P. (1984) Superior colliculus and visual neglect in rat and hamster. III. Functional implications. *Brain Research Reviews* 8: 155–63. [aBM]

Dean, P., Redgrave, P. & Westby, G. M. W. (1989) Event or emergency – 2 response systems in the mammalian superior colliculus. *Trends in Neurosciences* 12:137–47. [aBM]

DeCasper, A. J. & Fifer, W. P. (1980) Of human bonding: Newborns prefer their mothers' voices. *Science* 208:1174–76. [CI]

Delfour, F. & Marten, K. (2001) Mirror image processing in three marine mammal species: Killer whales (*Orcinus orca*), false killer whales (*Pseudorca crassidens*) and California sea lions (*Zalophus californianus*). *Behavioral Processes* 53:181–90. [EG]

Denham, S. A. & Burton, R. (2003) *Social and emotional prevention and intervention programming for preschoolers*. Kluwer Academic/Plenum. [CI]

Dennett, D. C. (1991) *Consciousness explained*. Little, Brown. [AM, EW]

Denny, A. P. & Behari, M. (1999) Motor fluctuations in Parkinson's disease. *Journal of the Neurological Sciences* 165:18–23. [DC]

Denny-Brown, D. (1962) The midbrain and motor integration. *Proceedings of the Royal Society of Medicine* 55:527–38. [aBM]

Denton, D. (2006) *The primordial emotions: The dawning of consciousness*. Oxford University Press. [JP]

Deransart, C., Le-Pham, B.-T., Hirsch, E., Marescaux, C. H., & Depaulis, A. (2001) Inhibition of the substantia nigra suppresses absences and clonic seizures in audiogenic rats, but not tonic seizures: Evidence for seizure specificity of the nigral control. *Neuroscience* 105:203–11. [aBM]

Derbyshire, S. W. G. (2006) Can fetuses feel pain? *British Medical Journal* 333:909–12. [KJSA]

Derbyshire, S. W. G. & Furedi, A. (1998) Do fetuses feel pain? "Fetal pain" is a misnomer. *British Medical Journal* 313(7060):795. [KJSA]

Devlin, A. M., Cross, J. H., Harkness, W., Chong, W. K., Harding, B., Varghu-Khadem, F. & Neville, B. C. R. (2003) Clinical outcomes of hemispherectomy for epilepsy in childhood and adolescence. *Brain* 126:556–66. [aBM]

Dommett, E., Coizet, V., Blaha, C. D., Martindale, J., Lefebvre, V., Walton, N., Mayhew, J. E., Overton, P. G. & Redgrave, P. (2005) How visual stimuli activate dopaminergic neurons at short latency. *Science* 307:1476–79. [FA]

Dorris, M. C. & Munoz, D. P. (1998) Saccadic probability influences motor preparation signals and time to saccadic initiation. *Journal of Neuroscience* 18(17):7015–26. [RJK]

Dostrovsky, J. O. (2000) Role of thalamus in pain. *Progress in Brain Research* 129:245–57. [MD]

Double, T. P., Skaliora, I., Baron, J. & King, A. J. (2003) Functional connectivity between the superficial and deeper layers of the superior colliculus: An anatomical substrate for sensorimotor integration. *The Journal of Neuroscience* 23:6596–607. [arBM]

Dretske, F. (1993) Conscious experience. *Mind* 102:1–21. [aBM]

Driver, J. & Vuilleumier, P. (2001) Perceptual awareness and its loss in unilateral neglect and extinction. *Cognition* 79:39–88. [aBM]

Durmer, J. S. & Rosenquist, A. C. (2001) Ibotenic acid lesions in the pedunculopontine region result in recovery of visual orienting in the hemianopic cat. *Neuroscience* 106:765–81. [aBM]

Eccles, J. C., ed. (1966) *Brain and conscious experience*. Springer Verlag. [aBM]

Edelman, D. B., Baars, B. J. & Seth, A. K. (2005) Identifying the hallmarks of consciousness in non-mammalian species. *Consciousness and Cognition* 14(1):169–87. [DC, DBE, AKS]

Edelman, G. M. (2003) Naturalizing consciousness: A theoretical framework. *Proceedings of the National Academy of Sciences USA* 100(9):5520–24. [AKS]

(2006) Second nature: The transformation of knowledge. In: *Second nature: Brain science and human knowledge*, ed. G. M. Edelman, pp. 142–57. Yale University Press. [CI]

Edelman, G. M. & Tononi, G. (2000) *A universe of consciousness. How matter becomes imagination*, 1st edition. Basic Books. [DBE, AKS]

Edwards, S. B. (1980) The deep layers of the superior colliculus: Their reticular characteristics and organization. In: *The reticular formation revisited*, ed. A. Hobson & M. A. B. Brazier, pp. 193–209. Raven Press. [arBM]

Edwards, S. B., Ginsburgh, C. L., Henkel, C. K. & Stein, B. E. (1979) Sources of subcortical projections to the superior colliculus in the cat. *Journal of Comparative Neurology* 184:309–29. [aBM]

Eibl-Eibesfeldt, I. (1975) *Ethology: The biology of behavior*, 2nd edition, trans. E. Klinghammer. Holt, Rinehart & Winston. [RBC]

Eimer, M., Hommel, B. & Prinz, W. (1995) S-R compatibility and response selection. *Acta Psychologica* 90:301–23. [EM]

Endo, T., Yanagawa, Y., Obata, K. & Isa, T. (2005) Nicotinic acetylcholine receptor subtypes involved in facilitation of CABAergic inhibition in mouse superficial superior colliculus. *Journal of Neurophysiology* 94:3893–902. [DC]

Engel, A. K., Fries, P., König, P., Brecht, M. & Singer, W. (1999) Temporal binding, binocular rivalry, and consciousness. *Consciousness and Cognition* 8: 128–51. [aBM]

Engel, A. K. & Singer, W. (2001) Temporal binding and the neural correlates of sensory awareness. *Trends in Cognitive Sciences* 5(1):16–25. [aBM, SMD]

Erickson, R. P. (1984) On the neural bases of behavior. *American Scientist* 351:233–41. [RAG]

Erickson, R. P., Di Lorenzo, P. M. & Woodbury, M. A. (1994) Classification of taste responses in the brain stem: Membership in fuzzy sets. *Journal of Neurophysiology* 71:2139–50. [RAG]

Erzurumlu, R. S. & Jhaveri, S. (1990) Thalamic axons confer a blueprint of the sensory periphery onto the developing rat somatosensory cortex. *Brain Research. Developmental Brain Research* 56(2):229–34. [KJSA]

Ewert, J.-P. (1968) Der Einfluss von Zwischenhirndefekten auf die Visuomotorik im Beutefang- und Fluchtverhalten der Erdkröte (*Bufo bufo L.*). *Zeitschrift für Vergleichende Physiologie* 61:41–70. [aBM]

Fecteau, J. H. & Munoz, D. P. (2006) Salience, relevance, and firing: A priority map for target selection. *Trends in Cognitive Sciences* 10:382–90. [aBM]

Feldt, K. S., Ryden, M. B. & Miles, S. (1998) Treatment of pain in cognitively impaired compared with cognitively intact older patients with hip fracture. *Journal of the American Geriatric Society* 46(6):1079–85. [KJSA]

Felleman, D. J. & Van Essen, D. C. (1991) Distributed hierarchical processing in the primate cerebral cortex. *Cerebral Cortex* 1:1–47. [aBM]

Fernandez de Molina, A. & Hunsperger, R. W. (1962) Organization of the subcortical system governing defense and flight reactions in the cat. *Journal of Physiology* 160:200–13. [aBM]

Ferrell, B. A., Ferrell, B. R. & Rivera, L. (1995) Pain in cognitively impaired nursing home patients. *Journal of Pain and Symptom Management* 10(8):591–98. [KJSA]

Fessard, A. E. (1954) Mechanisms of nervous integration and conscious experience. In: *Brain mechanisms and consciousness*, ed. E. D. Adrian, F. Bremer & H. H. Jasper, pp. 200–36. Blackwell. [aBM]

Ficalora, A. S. & Mize, R. R. (1989) The neurones of the substantia nigra and the zona incerta which project to the cat superior colliculus are GABA immunoreactive: A double label study using GABA immunocytochemistry and lectin retrograde transport. *Neuroscience* 29:567–81. [aBM]

Findlay, J. M. & Walker, R. (1999) A model of saccade generation based on parallel processing and competitive inhibition. *Behavioral and Brain Sciences* 22: 661–74. [aBM]

Fisk, N. M., Gian, R., Teixeira, J. M., Giannakoulopoulos, X., Cameron, A. D. & Glover, V. A. (2001) Effect of direct fetal opioid analgesia on fetal hormonal and hemodynamic stress response to intrauterine needling. *Anesthesiology* 95(4):828–35. [KJSA]

Fitzgerald, M. (2005) The development of nociceptive circuits. *Nature Reviews Neuroscience* 6(7):507–20. [KJSA]

Floyd, N. S., Price, J. L., Ferry, A. T., Keay, K. A. & Bandler, R. (2000) Orbitomedial prefrontal cortical projections to distinct longitudinal columns of the periaqueductal gray in the cat. *Journal of Comparative Neurology* 422: 556–78. [aBM]

Forster, M. C., Pardiwala, A. & Calthorpe, D. (2000) Analgesia requirements following hip fracture in the cognitively impaired. *Injury* 31(6):435–36. [KJSA]

Freeman, W. J. (2006) Definitions of state variables and state space for brain-computer interface. Part 1: Multiple hierarchical levels of brain function. *Cognitive Neurodynamics* 1(1):1871–3080 (print), 1871–4099 (Online). Available at: http://dx.doi.org/10.1007/s11571-006-9001-x [WJF]

Friede, R. L. (1989) *Developmental neuropathology*, pp. 31–39. Springer-Verlag. [aBM]

Friedman, D., Cycowicz, Y. M. & Gaeta, H. (2001) The novelty P3: An event-related brain potential (ERP) sign of the brain's evaluation of novelty. *Neuroscience and Biobehavioral Reviews* 25:355–73. [FB]

Fries, W. (1984) Cortical projections to the superior colliculus in the macaque monkey: A retrograde study using horseradish peroxidase. *Journal of Comparative Neurology* 230:55–76. [aBM]

Friston, K. (2005) A theory of cortical responses. *Philosophical Transactions of the Royal Society of London, B: Biological Sciences* 360:815–36. [FB]

Frisch, B., Sonntag, R., Dubuc, R., Ohta, Y. & Grillner, S. (1990) Organization of the six motor nuclei innervating the ocular muscles in lamprey. *Journal of Comparative Neurology* 294:491–506. [aBM]

Fujishiro, H., Umegaki, H., Isojima, D., Akatsu, H., Iguchi, A. & Kosaka, K. (2006) Depletion of cholinergic neurons in the nucleus of the medial septum and the vertical limb of the diagonal band in dementia with Lewy bodies. *Acta Neuropathologica (Berlin)* 111:103–14. [DC]

Fuster, J. M. (1997) *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe*. Lippincott-Raven. [FB]

Gallistel, C. R. (1999) Coordinate transformations in the genesis of directed action. In: *Cognitive science*, ed. B. O. M. Bly & D. E. Rumelhart, pp. 1–42. Academic Press. [aBM]

Gamper, E. (1926) Bau und Leistungen eines menschlichen Mittelhirnwesen (Arhinencephalie mit Encephalocele). *Zeitschrift für die gesamte Neurologie und Psychiatrie* 102:154–235, and 104: 49–120. [aBM]

Gardner, J. L. & Lisberger, S. G. (2002) Serial linkage of target selection for orienting and tracking eye movements. *Nature Neuroscience* 29:29. [RJK]

Gardner, R. A. & Gardner, B. T. (1998) *The structure of learning: From sign stimuli to sign language*. Erlbaum. [RAG]

Gawryszewski, L. G., Carreiro, L. R. R. & Magalhães, F. V. (2005) Early and late inhibitions elicited by a peripheral visual cue on manual response to a visual target: Are they based on Cartesian coordinates? *Psicológica* 26:121–37. [aBM]

Gaymard, B., Francois, C., Ploner, C. J., Condy, C. & Rivaud-Pechoux, S. (2003) A direct prefrontotectal tract against distractibility in the human brain. *Annals of Neurology* 53:542–45. [FB]

Gazzaniga, M. S. (2005) Forty-five years of split-brain research and still going strong. *National Review of Neuroscience* 6(8):653–59. [AM]

Gehring, W. J. (2005) New perspectives on eye development and the evolution of eyes and photoreceptors. *Journal of Heredity* 96:171–84. [DBE]

Geng, J. J. & Behrmann, M. (2006) Competition between simultaneous stimuli modulated by location probability in hemispatial neglect. *Neuropsychologia* 44:1050–60. [rBM]

Ghazanfar, A. A. & Schroeder, C. E. (2006) Is neocortex essentially multisensory? *Trends in Cognitive Science* 10:278–85. [EM]

Gibbs, F. A., Gibbs, E. L. & Lennox, W. G. (1937) Epilepsy: A paroxysmal cerebral dysrhythmia. *Brain* 60:377–88. [aBM]

Gibbs, F. A., Lennox, W. G. & Gibbs, E. L. (1936) Localizable features of the electrical activity of the brain in petit mal epilepsy. *American Journal of Physiology* 116:61. [aBM]

Gigerenzer, G., Todd, P. M. & the ABC Research Group (1999) *Simple heuristics that make us smart*. Oxford University Press. [RAG]

Gioanni, Y., Gioanni, H. & Mitrovic, N. (1991) Seizures can be triggered by stimulating non-cortical structures in the quaking mutant mouse. *Epilepsy Research* 9:19–31. [aBM]

Giolli, R. A., Gregory, K. M., Suzuki, D. A., Blanks, R. H., Lui, F. & Betelak, K. F. (2001) Cortical and subcortical afferents to the nucleus reticularis tegmenti pontis and basal pontine nuclei in the macaque monkey. *Visual Neuroscience* 18:725–40. [aBM]

Glassman, R. B. (1970) Cutaneous discrimination and motor control following somatosensory cortical ablations. *Physiology and Behavior* 5:1003–12. [RBG]

(1983) Dissociation of vertical and horizontal components of somesthetic orientation-localization during recovery from cortical damage: Implication regarding central associative functions. *Physiological Psychology* 11:47–53. [RBG]

(1985) Parsimony in neural representations: Generalization of a model of spatial orientation ability. *Physiological Psychology* 13:43–47. [RBG]

(1994) Behavioral specializations of SI and SII cortex: A comparative examination of the neural logic of touch in rats, cats, and other mammals. *Experimental Neurology* 125:134–41. [RBG]

(1999) A working memory "theory of relativity": Elasticity over temporal, spatial, and modality ranges conserves 7 ± 2 item capacity in radial maze, verbal tasks, and other cognition. *Brain Research Bulletin* 48:473–99. [RBG]

(2002) "Miles within millimeters" and other awe-inspiring facts about our "mortarboard" human cortex. *Zygon: Journal of Religion and Science* 37: 255–77. [RBG]

(2003). Hypothesized temporal and spatial code properties for a moment's working memory capacity: Brain wave "harmonics" and "four-color" topology of activated cortical areas. In: *Time and mind II: Information processing perspectives*, ed. H. Helfrich, pp. 161–83. Hogrefe & Huber. [RBG]

Glassman, R. B. & Smith, A. (1958) Neural spare capacity and the concept of diaschisis: Functional and evolutionary models. In: *Brain injury and recovery: Theoretical and controversial issues*, ed. S. Finger, T. LeVere, C. R. Almli & D. G. Stein, pp. 45–60. Plenum Press. [RBG]

Glenberg, A. M. (1997) What memory is for. *Behavioral and Brain Sciences* 20:1–55. [EM]

Glimcher, P. W. & Sparks, D. L. (1992) Movement selection in advance of action in the superior colliculus. *Nature* 355(6360):542–45. [FJK, aBM]

Glover, V. & Fisk, N. (1996) We don't know. Better to err on the safe side from mid-gestation. *British Medical Journal* 313:796. [KJSA]

Goldberg, H. M., II. & Malach, R. (2006) When the brain loses its self: Prefrontal inactivation during sensorimotor processing. *Neuron* 50:329–39. [AM]

Goldman-Rakic, P. S. & Nauta, W. J. (1976) Autoradiographic demonstration of a projection from prefrontal association cortex to the superior colliculus in the rhesus monkey. *Brain Research* 116:145–49. [FB]

Goldstein, L. B. & Simel, D. L. (2005) Is this patient having a stroke? *Journal of the American Medical Association* 293:2391–402. [DC]

Goltz, F. L. (1892) Der hund ohne grosshirn. Siebente Abhandlun, her die verrichtungen des grosshirns. *Pflügers Archiv* 51:570–614. [WJP]

Goodale, M. A. (1956) Visuomotor modules in the vertebrate brain. *Canadian Journal of Physiology and Pharmacology* 74:396–400. [rBM]

Goodale, M. A. & Murison, R. C. C. (1975) The effects of lesions of the superior colliculus on locomotor orientation and the orienting reflex in the rat. *Brain Research* 88:243–61. [aBM]

Gordon, N. S., Kollack-Walker, S., Akil, H. & Panksepp, J. (2002) Expression of c-fos gene activation during rough and tumble play in juvenile rats. *Brain Research Bulletin* 57:651–59. [aBM]

Goto, M., Canteras, N. S., Burns, G. & Swanson, L. W. (2005) Projections from the subfornical region of the lateral hypothalamic area. *Journal of Comparative Neurology* 493:412–38. [aBM]

Graf, W., Gerrits, N., Yatim-Dhiba, N. & Ugolini, G. (2002) Mapping the oculomotor system: The power of transneuronal labeling with rabies virus. *European Journal of Neuroscience* 15:1537–62. [aBM]

Grayhiel, A. M. (1978) A stereometric pattern of distribution of acetylcholinesterase in the deep layers of the superior colliculus. *Nature* 273: 539–41. [aBM]

Greenfield, S. A. (2000) *The private life of the brain*. Pergamon Press. [JvH]

Greenough, W. T. (1991) Experience as a component of normal development: Evolutionary considerations. *Developmental Psychology* 27:14–17. [CI]

Gregory, R. L. (1970) *The intelligent eye*. McGraw-Hill. [REG]

(1978) *Eye and brain*. 3rd edition. World University Library. [REG]

Grillner, S. (2003) The motor infrastructure: From ion channels to neuronal networks. *Nature Reviews, Neuroscience* 4:573–86. [aBM]

Grillner, S., Georgopoulos, A. P. & Jordan, L. M. (1997) Selection and initiation of motor behavior. In: *Neurons, networks, and motor behavior*, ed. P. S. G. Stein, S. Grillner, A. I. Selverson & D. G. Stuart, pp. 1–19. MIT Press. [aBM]

Grillner, S., Hellgren, J., Menard, A., Saitoh, K. & Wikstrom, M. A. (2005) Mechanisms for selection of basic motor programs – roles for the striatum and pallidum. *Trends in Neurosciences* 28:364–70. [rBM]

Grobstein, P. (1988) Between the retinotectal projection and directed movement: Topography of a sensorimotor interface. *Brain, Behavior and Evolution* 31:34–48. [aBM]

(1989) Organization in the sensorimotor interface: A case of increased resolution. In: *Visuomotor coordination*, ed. J.-P. Ewert & M. A. Arbib, pp. 537–68. Plenum Press. [aBM]

Groenewegen, H. J. (2003) The basal ganglia and motor control. *Neural Plasticity* 10:107–20. [aBM]

Grofova, I., Ottersen, O. P. & Rinvik, E. (1978) Mesencephalic and diencephalic afferents to the superior colliculus and periaqueductal gray substance demonstrated by retrograde axonal transport of horseradish peroxidase in the cat. *Brain Research* 146:205–20. [aBM]

Groh, J. M. & Sparks, D. L. (1996) Saccades to somatosensory targets. III. Eye-dependent somatosensory activity in primate superior colliculus. *Journal of Neurophysiology* 75:439–53. [aBM]

Grunau, R. V. & Craig, K. D. (1987) Pain expression in neonates: Facial action and cry. *Pain* 28(3):395–410. [KJSA]

Guillaume, A. & Pélisson, D. (2001) Gaze shifts evoked by electrical stimulation of the superior colliculus in the head-unrestrained cat. II. Effect of muscimol inactivation of the caudal fastigial nucleus. *European Journal of Neuroscience* 14:1345–59. [aBM]

Gurney, K., Prescott, T. J., Wickens, J. & Redgrave, P. (2004) Computational models of the basal ganglia: From membranes to robots. *Trends in Neurosciences* 27:453–59. [TJP]

Haber, S. N. & Fudge, J. L. (1997) The primate substantia nigra and VTA: Integrative circuitry and function. *Critical Reviews in Neurobiology* 11: 323–42. [aBM]

Haberly, L. B. (1998) Olfactory cortex. In: *The synaptic organization of the brain*. 4th edition, ed. G. M. Shepherd, pp. 377–416. Oxford University Press. [EM]

Haft, M. (1998) Robust "topological" codes by keeping control of internal redundancy. *Physical Review Letters* 81:4046–49. [aBM]

Halsey, J. (1987) Hydranencephaly. In: *Handbook of clinical neurology*, ed. P. Vinken, G. Bruyn & H. Klawans, pp. 337–53. Elsevier Science. [SW]

Hamani, C., Salabe, S., Bortoloto, Z. A., Cavalheiro, E. A. & Mello, L. (1984) Inhibitory role of the zona incerta in the pilocarpine model of epilepsy. *Epilepsy Research* 49:73–80. [aBM]

Hameroff, S. R. (2006) The entwined mysteries of anesthesia and consciousness. *Anesthesiology* 105:400–12. RRB]

Han, C. J., O'Tuathaigh, C. M., van Trigt, L., Quinn, J. J., Fanselow, M. S., Mongeau, R., Koch, C. & Anderson, D. J. (2003) Trace but not delay fear conditioning requires attention and the anterior cingulate cortex. *Proceedings of the National Academy of Sciences USA* 100:13087–92. [aBM]

Han, Z. Y., Zoli, M., Cardona, A., Bourgeois, J. P., Changeux, J. P. & Le Novere, N. (2003) Localization of ^{3}H]nicotine, ^{3}H]cytisine, ^{3}H]epibatidine, and ^{125}I]alpha-bungarotoxin binding sites in the brain of *Macaca mulatta*. *Journal of Comparative Neurology* 461:49–56. [DC]

Harding, D. E. (1961) *On having no head*. Arkana. [aBM]

Harland, D. P. & Jackson, R. R. (2000) "Eight-legged cats" and how they see: A review of recent research on jumping spiders (*Araneae Salticidae*). *Cimbebasia* 16:231–40. [EC]

Harting, J. K., Feig, S. & Van Lieshout, D. P. (1997) Cortical somatosensory and trigeminal inputs to the cat superior colliculus: Light and electron microscopic analyses. *Journal of Comparative Neurology* 388:313–26. [aBM]

Harting, J. K., Huerta, M. F., Frankfurter, A. J., Strominger, N. L. & Royce, G. J. (1980) Ascending pathways from the monkey superior colliculus: An autoradiographic analysis. *Journal of Comparative Neurology* 192: 853–82. [aBM]

Harting, J. K., Updyke, B. V. & Van Lieshout, D. P. (1992) Corticotectal projections in the cat: Anterograde transport studies of twenty-five cortical areas. *Journal of Comparative Neurology* 324:379–414. [aBM]

Hartline, P. H., Kass, L. & Loop, M. S. (1978) Merging of modalities in the optic tectum: Infrared and visual integration in rattlesnakes. *Science* 199:1225–29. [aBM]

Hassin, R. R., Uleman, J. S. & Bargh, J. A. (2005) *The new unconscious*. Oxford University Press. [CI]

Haynes, J. D., Deichmann, R. & Rees, G. (2005) Eye-specific effects of binocular rivalry in the human lateral geniculate nucleus. *Nature* 438(7067):496–99. [SW]

Heath, R. G. (1975) Brain function and behavior: I. Emotion and sensory phenomena in psychotic patients and in experimental animals. *Journal of Nervous and Mental Disease* 160:159–75. [aBM]

Hembree, E. A. (1986) Individual differences and developmental changes in infants' emotion expressions during early mother-infant interaction: Unpublished manuscript, University of Delaware. [CI]

Hepper, P. G. & Shahidullah, S. (1994) The beginnings of mind – evidence from the behavior of the fetus. *Journal of Reproductive and Infant Psychology* 12:143–54. [KJSA]

Hering, E. (1879/1942) *Spatial sense and movements of the eye*. trans. C. A. Radde. American Academy of Optometry. (Original work published in 1879). [aBM]

Herrick, C. J. (1948) *The brain of the tiger salamander*. University of Chicago Press. [WJP]

Hess, W. R. (1954) *Das Zwischenhirn. Syndrome, Lokalisationen, Funktionen*, 2nd edition. Benno Schwabe. aBM]

(1957) *The functional organization of the diencephalon*. Grune & Stratton. JP]

Hess, W. R. & Brugger, M (1943) Das subkortikale Zentrum der affektiven Abwehrreaktion. *Helvetica Physiologica Acta* 1:33–52. aBM]

Hess, W. R., Burgi, S. & Bucher, V. (1946) Motorische Funktion des tektal- und tegmentalgebietes. *Monatschift für Psychiatrie und Neurologie* 112: 1–52. aBM]

Hikosaka, O., Takikawa, Y. & Kawagoe, R. (2000) Role of the basal ganglia in the control of purposive saccadic eye movements. *Physiological Review* 80: 953–78. TJP]

Hikosaka, O. & Wurtz, R. H. (1983) Visual and oculomotor functions of monkey substantia nigra pars reticulata. I. Relation of visual and auditory responses to saccades. *Journal of Neurophysiology* 49:1230–53. Elsevier. [aBM]

(1989) The basal ganglia. In: *The neurobiology of saccadic eye movements*, ed. R. H. Wurtz & M. E. Goldberg, pp. 257–81. Elsevier. [aBM]

Hinson, J. M., Whitney, P., Holben, H. & Wirick, A. K. (2006) Affective biasing of choices in gambling task decision making. *Cognitive, Affective and Behavioral Neurosciences* 6:190–200. [JP]

Hirai, T., Onodera, S. & Kawamura, K. (1982) Cerebellotectal projections studied in cats with horseradish peroxidase or tritiated amino acids axonal transport. *Experimental Brain Research* 48:1–12. [aBM]

Hodgson, S. H. (1878) *The philosophy of reflection*. Longmans, Green. [aBM]

Hofer, M. A. (1994) Cardiac respiratory function during sudden prolonged immobility in wild rodents. *Psychosomatic Medicine* 32:633–47. [JvH]

Hoffman, J. & Kiss, L. (1969) "Hydranencephaly". A case report with autopsy findings in a 7-year-old girl. *Acta Paediatrica Scandinavica* 58: 297–300. [aBM]

Holland, P. W. H., García-Fernández, J., Williams, N. A. & Sidow, A. (1994) Gene duplication and the origins of vertebrate development. *Development* (Suppl.):125–33. aBM]

Holland, L. Z. & Holland, N. D. (1999) Chordate origins of the vertebrate central nervous system. *Current Opinion in Neurobiology* 9:596–602. aBM]

(2001) Evolution of neural crest and placodes: *Amphioxus* as a model for the ancestral vertebrate? *Journal of Anatomy* 199:85–98. [aBM]

Holsiege, J. C. (1991) Ultrastructural evidence for GABAergic brain stem projections to spinal motoneurons in the rat. *Journal of Neuroscience* 11: 159–67. aBM]

Holstege, G., Bandler, R. & Saper, C. B., eds. (1996) *The emotional motor system.* Elsevier. [aBM]

Holstege, G. & Cowie, R. J. (1989) Projections from the rostral mesencephalic reticular formation to the spinal cord. An HRP and autoradiographical tracing study in the cat. *Experimental Brain Research* 75:265–79. [aBM]

Holstege, G. & Georgiadis, J. R. (2004) The emotional brain: neural correlates of cat sexual behavior and human male ejaculation. *Progress in Brain Research* 143:39–45. [aBM]

Holzman, R. S. & Hickey, P. R. (2001) The development of pain perception and the stress response. In: *Pediatric anesthesia in the Harvard Electronic Anesthesia Library*, ed. M. T. Bailin. Lippincott Williams and Wilkins. [RRB]

Hommel, B., Müsseler, J., Aschersleben, G. & Prinz, W. (2001) The theory of event coding: A framework for perception and action planning. *Behavioral and Brain Sciences* 24:849–937. [EM]

Horgas, A. L. & Tsai, P. F. (1998) Analgesic drug prescription and use in cognitively impaired nursing home residents. *Nursing Research* 47(4):235–42. [KJSA]

Horowitz, G. D. & Newsome, W. T. (1999) Separate signals for target selection and movement specification in the superior colliculus. *Science* 284:1158–61. [aBM]

Horowitz, S. S., Blanchard, J. & Morin, L. P. (2005) Medial vestibular connections with the hypocretin (orexin) system. *Journal of Comparative Neurology* 487:127–46. [aBM]

Horvitz, J. C. (2000) Mesolimbocortical and nigrostriatal dopamine responses to salient non-reward events. *Neuroscience* 96:651–56. [aBM]

Houk, J. C. (1991) Red nucleus: Role in motor control. *Current Opinion in Neurobiology* 1:610–15. [aBM]

Hovda, D. A. & Villablanca, J. R. (1980) Sparing of visual field perception in neonatal but not adult cerebral hemispherectomized cats. Relationship with oxidative metabolism of the superior colliculus. *Behavioral Brain Research* 39:119–32. [aBM]

Howard, I. P. & Templeton, W. B. (1966) *Human spatial orientation.* Wiley. [aBM]

Humphries, M. D., Gurney, K. & Prescott, T. J. (2006) The brainstem reticular formation is a small-world, not scale-free, network. *Proceedings of the Royal Society, B. Biological Sciences* 273:503–11. [TJP]

(In press) Is there a brainstem substrate for action selection? *Philosophical Transactions of the Royal Society of London, Series B. Biological Sciences.* [TJP, AKS]

Hunsperger, R. W. (1956) Affektreaktionen auf elektrische reizung im Hirnstamm der Katze. *Helvetica Physiologica Acta* 14:70–92. [aBM]

(1963) Comportements affectifs provoqués par la stimulation électrique du tronc cérébral et du cerveau antérieur. *Journal de Physiologie* 55:45–97. [aBM]

Hunsperger, R. W. & Bucher, V. M. (1967) Affective behaviour produced by electrical stimulation in the forebrain and brainstem of the cat. *Progress in Brain Research* 27:103–27. [aBM]

Husserl, E. (1991) *On the phenomenology of the consciousness of internal time (1893–1917)*; trans. J. Barnett Brough. Kluwer Academic. [GN]

Huston, J. P. & Tomaz, C. (1996) Subtelencephalic locale of reinforcement and learning: looking for the minimal necessary structures. *Behavioural Brain Research* 22:153–61. [aBM]

Hyde, P. S. & Knudsen, E. I. (2001) A topographic instructive signal guides the adjustment of the auditory space map in the optic tectum. *Journal of Neuroscience* 21:8586–93. [rBM]

Hydranencephaly Group Survey (2003) Unpublished questionnaire survey of 81 families conducted by Barb Aleman from September 2002 through February 2003. Available at: http://hydranencephaly.com/researchresults.htm [aBM]

Iacono, R. P. & Nashold, B. S., Jr. (1952) Mental and behavioral effects of brain stem and hypothalamic stimulation in man. *Human Neurobiology* 1: 273–79. [aBM]

Illing, R.-B. (1992) Association of efferent neurons to the compartmental architecture of the superior colliculus. *Proceedings of the National Academy of Sciences USA* 89:10900–904. [aBM]

Illing, R.-B. & Graybiel, A. M. (1986) Complementary and non-matching afferent compartments in the cat's superior colliculus: Innervation of the acetylcholinesterase-poor domain of the intermediate gray layer. *Neuroscience* 18: 373–94. [aBM]

Ingle, D. J. (1974) Two visual systems in the frog. *Science* 181:1053–55. [aBM]

(1991) Functions of subcortical visual systems in vertebrates and the evolution of higher visual mechanisms. In: *Evolution of the eye and visual system*, ed. J. R. Cronly-Dillon & R. L. Gregory, pp. 152–64. CRC Press. [rBM]

Isa, T., Endo, T. & Saito, Y. (1998) The visuo-motor pathway in the local circuit of the rat superior colliculus. *Journal of Neuroscience* 18:8486–504. [rBM]

Isa, T. & Kobayashi, Y. (2004) Switching between cortical and subcortical sensorimotor pathways. *Progress in Brain Research* 143:299–305. [aBM]

Isa, T. & Sasaki, S. (2002) Brainstem control of head movements during orienting: Organization of the premotor circuits. *Progress in Neurobiology* 66: 205–41. [aBM]

Iwahori, N., Kawawaki, T. & Baba, J. (1999) Neuronal organization of the optic tectum in the river lamprey, *Lampetra japonica*: A Golgi study. *Journal für Hirnforschung* 39:409–24. [aBM]

Izard, C. E. (1990) The substrates and functions of emotions feelings: William James and current emotion theory. *Personality and Social Psychology Bulletin* 16:626–35. [CI]

(1991) *The psychology of emotions.* Plenum Press. [CI]

(2002) Translating emotion theory and research into preventive interventions. *Psychological Bulletin* 128:796–824. [CI]

(2007) Basic emotions, natural kinds, emotion schemas, and a new paradigm. Unpublished manuscript. [CI]

Izard, C. E., Fantauzzo, C. A., Castle, J. M., Haynes, O. M., Rayias, M. F. & Putnam, P. H. (1995) The ontogeny and significance of infants' facial expressions in the first 9 months of life. *Developmental Psychology* 31: 997–1013. [CI]

Izard, C. E., Hembree, E. A. & Huebner, R. R. (1987) Infants' emotion expressions to acute pain: Developmental change and stability of individual differences. *Developmental Psychology* 23:105–13. [CI]

Jackson, J. H. (1958) Evolution and dissolution of the nervous system. In: *Selected writings of John Hughlings Jackson*, ed. J. Taylor, pp. 45–118. Staples Press. [JvH]

Jackson, S. R., Newport, R., Mort, D. & Husain, M. (2005) Where the eye looks, the hand follows: Limb-dependent magnetic misreaching in optic ataxia. *Current Biology* 15:42–46. [aBM]

Jacobs, L. F. (1994) Natural space-use patterns and hippocampal size in kangaroo rats. *Brain, Behavior and Evolution* 44:125–32. [WJH]

James, W. (1890) *The principles of psychology, vol. I.* Dover. [R-PB]

(1890/1950) *The principles of psychology, Vol. 2.* Dover. [CI]

(1890/1983) *The principles of psychology*, with introduction by G. A. Miller. Harvard University Press. [aBM]

Jasper, H. H. & Drooglever-Fortuyn, J. (1947) Experimental studies on the functional anatomy of petit mal epilepsy. *Proceedings of the Association for Research in Nervous and Mental Disease* 26:272–98. [aBM]

Jasper, H. H., Proctor, L. D., Knighton, R. S., Noshay, W. C. & Costello, R. T., eds. (1958) *Reticular formation of the brain. Henry Ford Hospital International Symposium.* Little, Brown. [aBM]

Jay, M. F. & Sparks, D. L. (1987) Sensorimotor integration in the primate superior colliculus. II. Coordinates of auditory signals. *Journal of Neurophysiology* 57:35–55. [aBM]

Jennett, B. (2002) *The vegetative state. Medical facts, ethical and legal dilemmas.* Cambridge University Press. [aBM]

Jesberger, J. A. & Richardson, J. S. (1985) Animal models of depression: Parallels and correlates to severe depression in humans. *Biological Psychiatry* 20: 764–84. [WJH]

Jiang, H., Stein, B. E. & McHaffie, J. G. (2003) Opposing basal ganglia processes shape midbrain visuomotor activity bilaterally. *Nature* 423:982–86. [aBM]

Johansson, R. S., Westling, G., Bäckström, A. & Flanagan, J. R. (2001) Eye-hand coordination in object manipulation. *Journal of Neuroscience* 21: 6917–32. [aBM]

Johnson, S. C., Bader, L. C., Wilder, L. S., Piper, J. C., Heiserman, J. E. & Prigatano, G. P. (2002) Neural correlates of self-reflection. *Brain* 125: 1808–14. [AM]

Johnston, C. C., Collinge, J. M., Henderson, S. J. & Anand, K. J. S. (1997) A cross-sectional survey of pain and pharmacological analgesia in Canadian neonatal intensive care units. *Clinical Journal of Pain* 13(4):308–12. [KJSA]

Jones, E. G. (1998) A new view of specific and nonspecific thalamocortical connections. In: *Consciousness: At the frontiers of neuroscience*, ed. H. H. Jasper, L. Descarries, V. F. Castellucci & S. Rossignol. In Series: *Advances in Neurology* 77:49–71. Elsevier. [aBM]

(2002) Thalamic circuitry and thalamocortical synchrony. *Philosophical Transactions of the Royal Society, London, Series B* 357:1659–73. [aBM]

Jones, R. M. & France, T. D. (1978) Electrophysiological responses in hydranencephaly. *American Journal of Ophthalmology* 85:478–84. [aBM]

Jordan, L. M. (1998) Initiation of locomotion in mammals. *Annals of the New York Academy of Sciences* 860:83–93. [aBM]

Julesz, B. (1971) *Foundations of cyclopean perception.* University of Chicago Press. [aBM]

Jurgens, U. (1994) The role of the periaqueductal grey in vocal behaviour. *Behavioural Brain Research* 62:107–17. [aBM]

Kaada, B. R. (1951) Somato-motor, autonomic and electrocorticographic responses to electrical stimulation of rhinencephalic and other structures in primates, cat, and dog: A study of responses from the limbic, subcallosal, orbito-insular, piriform and temporal cortex, hippocampus-fornix and amygdala. *Acta Physiologica Scandinavica (Suppl.)* 24:1–262. [aBM]

Karten, H. J. (1997) Evolutionary developmental biology meets the brain: The origins of mammalian cortex (commentary). *Proceedings of the National Academy of Sciences USA* 94:2800–804. [DBE]

Kawamura, K. & Konno, T. (1979) Various types of corticotectal neurons of cats as demonstrated by means of retrograde axonal transport of horseradish peroxidase. *Experimental Brain Research* 35:161–75. [aBM]

Kawato, M., Hayakawa, H. & Inui, T. (1993) A forward-inverse optics model of reciprocal connections between visual cortical areas. *Network: Computation in Neural Systems* 4:415–22. [aBM]

Keay, K. A. & Bandler, R. (2002) Distinct central representations of inescapable and escapable pain: Observations and speculation. *Experimental Physiology* 87:275–79. [aBM]

Keenan, J. P., Wheeler, M. A., Gallup, Jr., G. G. & Pascual-Leone, A. (2000) Self-recognition and the right prefrontal cortex. *Trends in Cognitive Sciences* 4:338–44. [EG]

Keller, E. L., Lee, K.-M. & McPeek, R. M. (2005) Readout of higher-level processing in the discharge of superior colliculus neurons. *Annals of the New York Academy of Sciences* 1039:1–11. [aBM]

Kendrick, K. M., Hinton, M. R. & Baldwin, B. A. (1991) GABA release in the zona incerta of the sheep in response to the sight and ingestion of food and salt. *Brain Research* 550:165–68. [aBM]

Kim, U., Gregory, E. & Hall, W. C. (1992) Pathway from the zona incerta to the superior colliculus in the rat. *Journal of Comparative Neurology* 321: 555–75. [aBM]

Kiperstok, O. & Patterson, J. C. (1995) Intelligent fuzzy control to augment the scheduling capabilities of network queuing systems. *Computer Science Lecture Notes, vol. 949*. Springer-Verlag. [RAG]

Kittelberger, J. M., Land, B. R. & Bass, A. H. (2006) The midbrain periaqueductal gray and vocal patterning in a teleost fish. *Journal of Neurophysiology* 96: 71–85. [aBM]

Kjaer, T. W., Novak, M. & Lou, H. C. (2002) Reflective self-awareness and conscious states: PET evidence for a common midline parietofrontal core. *NeuroImage* 17:1080–86. [AM]

Klier, E. M., Wang, H. & Crawford, J. D. (2001) The superior colliculus encodes gaze commands in retinal coordinates. *Nature Neuroscience* 4:627–32. [aBM]

Knight, R. T. & Scabini, D. (1998) Anatomic bases of event-related potentials and their relationship to novelty detection in humans. *Journal of Clinical Neurophysiology* 15:3–13. [TB]

Koch, C. & Tsuchiya, N. (2007) Attention and consciousness: Two distinct brain processes. *Trends in Cognitive Science* 11:16–22. [FA]

Koh, J. L., Fanurik, D., Harrison, D. D., Schmitz, M. L. & Norvell, D. (2004) Analgesia following surgery in children with and without cognitive impairment. *Pain* 111(3):239–244. [KJSA]

Kolb, B. & Tees, C., eds. (2000) *The cerebral cortex of the rat*. MIT Press. [JP]

Kolmac, C. I., Power, B. D. & Mitrofanis, J. (1998) Patterns of connections between zona incerta and brainstem in rats. *Journal of Comparative Neurology* 396:544–55. [aBM]

König, P., Engel, A. K. & Singer, W. (1996) Integrator or coincidence detector? The role of the cortical neuron revisited. *Trends in Neurosciences* 19: 130–37. [aBM]

Körding, K. P. & Wolpert, D. M. (2006) Bayesian decision theory in sensorimotor control. *Trends in Cognitive Sciences* 10:319–26. [aBM]

Kosko, B. (1993) *Fuzzy thinking: The new science of fuzzy logic*. Hyperion. [RAC]

Kostovic, I. & Rakic, P. (1990) Developmental history of the transient subplate zone in the visual and somatosensory cortex of the macaque monkey and human brain. *Journal of Comparative Neurology* 297:441–70. [RRB]

Kozloski, J., Hamzei-Sichani, F. & Yuste R. (2001) Stereotyped position of local synaptic targets in neocortex. *Science* 293:868–72. [aBM]

Krauzlis, R. J. (2001) Extraretinal inputs to neurons in the rostral superior colliculus of the monkey during smooth-pursuit eye movements. *Journal of Neurophysiology* 86:2629–33. [aBM]

Krauzlis, R. J., Basso, M. A. & Wurtz, R. H. (1997) Shared motor error for multiple eye movements. *Science* 276(5319):1693–95. [RJK]

(2000) Discharge properties of neurons in the rostral superior colliculus of the monkey during smooth-pursuit eye movements. *Journal of Neurophysiology* 84(2):876–91. [RJK]

Krauzlis, R. J. & Dill, N. (2002) Neural correlates of target choice for pursuit and saccades in the primate superior colliculus. *Neuron* 35(2):355–63. [RJK]

Krauzlis, R. J., Liston, D. & Carello, C. D. (2004) Target selection and the superior colliculus: Goals, choices and hypotheses. *Vision Research* 44: 1445–51. [aBM]

Kuypers, H. G. J. M. (1982) A new look at the organization of the motor system. *Progress in Brain Research* 57:381–403. [aBM]

(1987) Some aspects of the organization of the output of the motor cortex. In: *Motor areas of the cerebral cortex, Ciba Foundation Symposium, 132*, ed. C. Bock, M. O'Connor & J. Marsh, pp. 63–82. Wiley. [aBM]

Kuypers, H. G. J. M. & Martin, G. F., eds. (1982) *Anatomy of descending pathways to the spinal cord. Progress in Brain Research, vol. 57*. Elsevier Biomedical. [aBM]

Lacalli, T. C. (1996) Landmarks and subdomains in the larval brain of *Branchiostoma*: Vertebrate homologs and invertebrate antecedents. *Israel Journal of Zoology* 42:S151–46. [aBM]

(2001) New perspectives on the evolution of protochordate sensory and locomotory systems, and the origin of brains and heads. *Philosophical Transactions of the Royal Society of London, B* 356:1565–72. [aBM]

Lacmle. L. K. (1953) A Golgi study of cell morphology in the deep layers of the human superior colliculus. *Journal für Hirnforschung* 24:297–306. [aBM]

Lambie, J. A. & A. J. Marcel (2002) Consciousness and the varieties of emotion experience: A theoretical framework. *Psychological Review* 109(2): 219–59. [GN]

Lamme, V. A. F. & Spekreijse, H. (2000) Modulations of primary visual cortex activity represent attentive and conscious scene perception. *Frontiers in Bioscience* 5:232–43. [aBM]

Land, M. F. (1974) A comparison of the visual behavior of a predatory arthropod with that of a mammal. In: *Invertebrate neurons and behavior*, ed. C. A. G. Wiersma, pp. 341–43. MIT Press. (Originally published in *The Neurosciences: Third Study Program*, pp. 411–18, same year). [FG]

Lang, C. E. & Schieber, M. H. (2003) Differential impairment of individuated finger movements in humans after damage to the motor cortex or the corticospinal tract. *Journal of Neurophysiology* 90:1160–70. [aBM]

Langer, T. P. & Lund, R. D. (1974) The upper layers of the superior colliculus of the rat: A Golgi study. *Journal of Comparative Neurology* 158:418–35. [aBM]

Larkum, M. E., Senn, W. & Lüscher, H.-R. (2004) Top-down dendritic input increases the gain of layer 5 pyramidal neurons. *Cerebral Cortex* 14: 1059–70. [aBM]

Lavallée, P., Urbain, N., Dufresne, C., Bokor, H., Acsady, L. & Deschenes, M. (2005) Feedforward inhibitory control of sensory information in higher-order thalamic nuclei. *Journal of Neuroscience* 25:7489–98. [aBM]

Lawrence, D. G. & Kuypers, H. G. J. M. (1968) The functional organization of the motor system in the monkey. I. The effects of bilateral pyramidal lesions. *Brain* 91:1–14. [aBM]

LeDoux, J. E. (1996) *The emotional brain: The mysterious underpinnings of emotional life*. Simon & Schuster. [CI, EM]

(2002) *The synaptic self*. Viking. [JvII]

Lee, H. W., Hong, S. B., Seo, D. W., Tae, W. S. & Hong, S. C. (2000) Mapping of functional organization in human visual cortex: Electrical cortical stimulation. *Neurology* 54(4):849–54. [SW]

Lee, P. H., Helms, M. C., Augustine, G. J. & Hall, W. C. (1997) Role of intrinsic synaptic circuitry in collicular sensorimotor integration. *Proceedings of the National Academy of Sciences USA* 94:13299–304. [aBM]

Lee, S. J., Ralston, H. J. P., Drey, E. A., Partridge, J. C. & Rosen, M. A. (2005) Fetal pain: A systematic multidisciplinary review of the evidence. *Journal of the American Medical Association* 294(8):947–54. [KJSA]

Leh, S. E., Johansen-Berg, H. & Ptito, A. (2006) Unconscious vision: New insights into the neuronal correlate of blindsight using diffusion tractography. *Brain* 129(Pt 7):1822–32. [SMD, SW]

Lehar, S. (2002) *The world in your head: A gestalt view of the mechanism of conscious experience*. Erlbaum. [aBM]

Leichnetz, G. R., Gonzalo-Ruiz, A., DeSalles, A. A. & Hayes, R. L. (1987) The origin of brainstem afferents of the paramedian pontine reticular formation in the cat. *Brain Research* 422:389–97. [aBM]

Lewis, M. Sullivan, M. W. & Michalson, L. (1983) The cognitive-emotional fugue. In: *Emotions, cognition, and behavior*, ed. C. E. Izard, J. Kagan & R. Zajonc, pp. 264–88. Cambridge University Press. [CI]

Libet, B. (1973) Electrical stimulation of cortex in human subjects, and conscious sensory aspects. In: *Handbook of sensory physiology, vol. II*, ed. A. Iggo, pp. 743–90. Springer-Verlag. [MD]

(1956) Unconscious cerebral initiative and the role of conscious will in voluntary action. *Behavioral and Brain Sciences* 8:529–66. [EM]

(2005) *Mind time: The temporal factor in consciousness*. Harvard University Press. [EM]

Libet, B., Wright, E. W., Jr., Feinstein, B. & Pearl, D. K. (1979) Subjective referral of the timing for a conscious sensory experience: A functional role for the somatosensory specific projection system in man. *Brain* 102:193–224. [rBM]

Lippa, C. F., Smith, T. W. & Perry, E. (1999) Dementia with Lewy bodies: Choline acetyltransferase parallels nucleus basalis pathology. *Journal of Neural Transmission* 106:525–35. [DC]

Liston, D. & Krauzlis, R. J. (2005) Shared response preparation for pursuit and saccadic eye movements. *Journal of Neuroscience* 25(38):11305–14. [RJK]

(2005) Shared decision signal explains performance and timing of pursuit and saccadic eye movements. *Journal of Vision* 5(9): 678–89. [RJK]

Llinas, R. R. & Pare, D. (1991) Of dreaming and wakefulness. *Neuroscience* 44:521–35. [R-PB]

Llinas, R. R. & Ribary, U. (1993) Coherent 40-Hz oscillation characterizes dream state in humans. *Proceedings of the National Academy of Sciences USA* 90:2078–81. [R-PB]

Lloyd-Thomas, A. R. & Fitzgerald, M. (1986) Do fetuses feel pain? Reflex responses do not necessarily signify pain. *British Medical Journal* 313(7060):797–98. [KJSA]

Lomber, S. G. & Payne, B. R. (1996) Removal of two halves restores the whole: Reversal of visual hemineglect during bilateral cortical and collicular inactivation in the cat. *Visual Neuroscience* 13:1143–56. [aBM]

Lomber, S. G., Payne, B. R. & Cornwell, P. (2001) Role of the superior colliculus in analyses of space: Superficial and intermediate layer contributions to visual orienting, auditory orienting, and visuospatial discriminations during unilateral and bilateral deactivations. *Journal of Comparative Neurology* 441:44–57. [aBM]

Lonstein, J. S., Simmons, D. A. & Stern, J. M. (1998) Functions of the caudal periaqueductal gray in lactating rats: kyphosis, lordosis, maternal aggression and fearfulness. *Behavioral Neuroscience* 112:1502–18. [aBM]

Loop, M. S. & Sherman, S. M. (1977) Visual discriminations of cats with cortical and tectal lesions. *Journal of Comparative Neurology* 174:79–88. [SMD, aBM]

Lotz, L. T., McPherson, D. L. & Starr, A. (1986) Cerebral cortical contributions to sensory evoked potentials: Hydranencephaly. *Electroencephalography and Clinical Neurophysiology* 64:218–23. [aBM]

Lundqvist, D. & Ohman, A. (2005) Emotion regulates attention: The relation between facial configurations, facial emotion, and visual attention. *Visual Cognition* 12:51–84. [CI]

Lynch, J. C., Hoover, J. E. & Strick, P. L. (1994) Input to the primate frontal eye field from the substantia nigra, superior colliculus, and dentate nucleus demonstrated by transneuronal transport. *Experimental Brain Research* 100:181–86. [aBM]

Lyon, D. C., Nassi, J. J. & Callaway, E. M. (2005) Disynaptic connections from the superior colliculus to cortical area MT revealed through transynaptic labeling with rabies virus (Abstract). *Journal of Vision* 5:432a. Available at: http://journalofvision.org/5/8/432/ [aBM]

Ma, T. P. (1996) Saccade-related omnivectoral pause neurons in the primate zona incerta. *Neuroreport* 7:2713–16. [aBM]

Ma, T. P., Cheng, H. W., Czoch, J. A. & Rafols, J. A. (1990) Intermediate and deep layers of the macaque superior colliculus: A Golgi study. *Journal of Comparative Neurology* 295:92–110. [aBM]

MacLean, P. D. (1990) *The triune brain in evolution: Role in paleocerebral functions*. Plenum Press. [JF, JvH]

Maes, P. (1990) Situated agents can have goals. In: *Designing autonomous agents*, ed. P. Maes, pp. 49–70. MIT Press. [aBM]

Magoun, H. W. (1954) The ascending reticular system and wakefulness. In: *Brain mechanisms and consciousness*, ed. E. D. Adrian, F. Bremer & H. H. Jasper, pp. 1–20. Blackwell. [aBM]

Malviya, S., Voepel-Lewis, T., Tait, A., Merkel, S., Lauer, A., Munro, H., Farley, F. (2001) Pain Management in children with and without cognitive impairment following spine fusion surgery. *Paediatric Anaesthesia* 11(4):453. [KJSA]

Mana, S. & Chevalier, G. (2002) The fine organization of nigro-collicular channels with additional observations of their relationships with acetylcholinesterase in the rat. *Neuroscience* 106:357–74. [aBM]

Mandler, G. A. (1975) Consciousness: Respectable, useful and *probably* necessary. In: *Information processing and cognition: The Loyola Symposium*, ed. R. Solso, pp. 229–54. Erlbaum. [aBM, AM]

(2002) *Consciousness recovered. Psychological functions and origins of conscious thought*. John Benjamins. [aBM]

Manger, P. R. (2006) An examination of cetacean brain structure with a novel hypothesis correlating thermogenesis to the evolution of a big brain. *Biological Review* 81:293–338. [EC]

Marin-Padilla, M. (1997) Developmental neuropathology and impact of perinatal brain damage. II: White matter lesions of the neocortex. *Journal of Neuropathology and Experimental Neurology* 56:219–35. [aBM]

Mashour, G. A. (2006) Integrating the science of consciousness and anesthesia. *Anesthesia and Analgesia* 103:975–82. [DC]

Masino, T. (1992) Brainstem control of orienting movements: Intrinsic coordinate system and underlying circuitry. *Brain, Behavior and Evolution* 40:98–111. [aBM]

Masino, T. & Grobstein, P. (1989) The organization of descending tectofugal pathways underlying orienting in the frog, Rana pipiens. I. Lateralization, parcellation, and an intermediate spatial representation. *Experimental Brain Research* 75:227–44. [aBM]

Masino, T. & Knudsen, E. I. (1990) Horizontal and vertical components of head movement are controlled by distinct neural circuits in the barn owl. *Nature* 345:434–37. [aBM]

Maskos, U., Molles, B. E., Pons, S., Besson, M., Guiard, B. P., Guilloux, J. P., Evrard, A., Cazala, P., Cormier, A., Mameli-Engvall, M., Dufour, N., Cloez-Tayarani, I., Bemelmans, A. P., Mallet, J., Gardier, A. M., David, V., Faure, P., Granon, S. & Changeux, J. P. (2005) Nicotine reinforcement and cognition restored by targeted expression of nicotinic receptors. *Nature* 436:103–107. [aBM]

Mather, J. A. (in press) Cephalopod consciousness: Behavioural evidence. *Consciousness and Cognition*. [DBE]

May, P. J. (2005) The mammalian superior colliculus: Laminar structure and connections. *Progress in Brain Research* 151:321–78. [aBM]

May, P. J., Hartwich-Young, R., Nelson, J., Sparks, D. L. & Porter, J. D. (1990) Cerebellotectal pathways in the macaque: Implications for collicular generation of saccades. *Neuroscience* 36:305–24. [aBM]

May, P. J., Sun, W. & Hall, W. C. (1997) Reciprocal connections between the zona incerta and the pretectum and superior colliculus of the cat. *Neuroscience* 77:1091–14. [aBM]

Mazzola, L., Isnard, J. & Mauguiere, F. (2006) Somatosensory and pain responses to stimulation of the second somatosensory area (SII) in humans: A comparison with SI and insular responses. *Cerebral Cortex* 16:960–68. [MD]

McAbee, G. N., Chan, A. & Erde, E. L. (2000) Prolonged survival with hydranencephaly: Report of two patients and literature review. *Pediatric Neurology* 23(1):80–84. [aBM, SW]

McBride, R. (1999) Consciousness and the state/transitive/creature distinction. *Philosophical Psychology* 12:181–96. [rBM]

McCormick, D. A. & Contreras, D. (2001) On the cellular and network bases of epileptic seizures. *Annual Review of Physiology* 63:815–46. [aBM]

McFarland, D. (1965) Flow graph representation of motivational systems. *British Journal of Mathematical and Statistical Psychology* 18:25–43. [aBM]

McFarland, D. J. & Houston, A. (1981) *Quantitative ethology: The state space approach*. Pitman. [aBM]

McFarland, D. J. & Sibly, R. M. (1975) The behavioural final common path. *Philosophical Transactions of the Royal Society (London)* 270:265–93. [aBM]

McGurk, H. & MacDonald, J. (1976) Hearing lips and seeing voices. *Nature* 264:746–48. [EM]

McHaffie, J. G., Kao, C. Q. & Stein, B. E. (1989) Nociceptive neurons in rat superior colliculus: Response properties, topography, and functional implications. *Journal of Neurophysiology* 62:510–25. [rBM]

McHaffie, J. G., Norita, M., Durning, D. D. & Stein, B. E. (1993) Corticotectal relationships: Direct and "indirect" pathways. *Progress in Brain Research* 95:139–50. [aBM]

McHaffie, J. G., Stanford, T. R., Stein, B. E., Coizet, V. & Redgrave, P. (2005) Subcortical loops through the basal ganglia. *Trends in Neurosciences* 28: 401–407. [TJP]

McPeek, R. M. & Keller, E. L. (2002) Saccade target selection in the superior colliculus during a visual search task. *Journal of Neurophysiology* 88(4): 2019–34. [RJK]

(2004) Deficits in saccade target selection after inactivation of the superior colliculus. *Nature Neuroscience* 7(7):757–63. [RJK, aBM]

Medina, L. & Reiner, A. (2000) Do birds possess homologues of primary visual, somatosensory and motor cortices? *Trends in Neurosciences* 23:1–12. [DBE]

Meeren, H., van Luijtelaar, G., Lopes da Silva, F. & Coenen, A. (2005) Evolving concepts on the pathophysiology of absence seizures: The cortical focus theory. *Archives of Neurology* 62:371–76. [AMLC, arBM]

Mellor, D. J., Diesch, T. J., Gunn, A. J. & Bennet, L. (2005). The importance of 'awareness' for understanding fetal pain. *Brain Research Reviews* 49: 455–71. [KJSA]

Melzack, R. & Wall, P. D. (1965) Pain mechanisms: A new theory. *Science* 150(699):971–79. [KJSA]

Menzel, C. (2005) Progress in the study of chimpanzee recall and episodic memory. In: *The missing link in cognition: Origins of self-reflective consciousness*, ed. H. S. Terrace & J. Metcalfe, pp. 188–224. Oxford University Press. [AM]

Meredith, M. A. & King, A. J. (2004) Spatial distribution of functional superficial–deep connections in the adult ferret superior colliculus. *Neuroscience* 128:861–70. [aBM]

Merculdi, M. A. & Ramos, A. S. (1998) Intrinsic circuitry of the superior colliculus: Pharmacophysiological identification of horizontally oriented inhibitory interneurons. *Journal of Neurophysiology* 79:1597–602. [aBM]

Merker, B. (1980) *The sentinel hypothesis: A role for the mammalian superior colliculus*. Doctoral dissertation. Department of Psychology and Brain Science, Massachusetts Institute of Technology. [aBM]

(1997) The common denominator of conscious states: Implications for the biology of consciousness. *Cogprints Electronic Archive. Biology: Theoretical biology*. [Archives preprint.] Available at: (http://cogprints.soton.ac.uk) [arBM]

(2004a) Cortex, countercurrent context, and dimensional integration of lifetime memory. *Cortex* 40:559–76. [arBM]

(2004b) The local and the global in cortical phylogeny and function. Reply to Finlay and Lamme. *Cortex* 40:582–83. [aBM]

(2005) The liabilities of mobility: A selection pressure for the transition to cortex in animal evolution. *Consciousness and Cognition* 14:89–114. [arBM]

Merskey, H. & Bogduk, N., eds. (1994) *Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms*, pp. 209–214. IASP Press. [KJSA]

Mesulam, M.-M. (1999) Spatial attention and neglect: Parietal, frontal and cingulate contributions to the mental representation and attentional targeting of salient extrapersonal events. *Philosophical Transactions of the Royal Society of London, B* 354:1325–46. [aBM]

Meyer, J.-A. & Wilson, S. W., eds. (1991) *From animals to animats: Proceedings of the First International Conference on Simulation of Adaptive Behavior*. MIT Press/Bradford Books. [aBM]

Miller, E. K. & Cohen, J. D. (2001) An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience* 24:167–202. [FB]

Miller, G. A. (1988) Interview with George A. Miller. In: *The cognitive revolution in psychology*, ed. B. J. Baars, pp. 199–222. Guilford Press. [aBM, AM]

Milner, B. (1968) Amnesia following operation on the temporal lobes In: *Amnesia*, ed. C. W. M. Whitty & O. L. Zangwill, pp. 109–33. Butterworths. [EM]

Milner, P. M. (1970) *Physiological psychology*. Holt, Rinehart & Winston. [RBG]

Mitrofanis, J. (2005) Some certainty for the "zone of uncertainty"? Exploring the function of the zona incerta. *Neuroscience* 130:1–15. [arlM, TJP]

Mitrofanis, J. & Mikuletic, L. (1999) Organization of the cortical projection to the zona incerta of the thalamus. *Journal of Comparative Neurology* 412: 173–185. [aBM]

Mize, R. R., Whitworth, H., Nunes-Cardozo, B. & van der Want, J. (1994) Ultrastructural organization of GABA in the rabbit superior colliculus revealed by quantitative postembedding immunocytochemistry. *Journal of Comparative Neurology* 341:273–87. [aBM]

Montague, D. P. F. & Walker-Andrews, A. S. (2001) Peekaboo: A new look at infants' perception of emotion expressions. *Developmental Psychology* 37:826–38. [CI]

Montague, P. R., Hyman, S. E. & Cohen, J. D. (2004) Computational roles for dopamine in behavioural control. *Nature* 431:760–67. [FA]

Morcuende, S., Delgado-Garcia, J.-M. & Ugolini, G. (2002) Neuronal premotor networks involved in eyelid responses: Retrograde transneuronal tracing with rabies virus from the orbicularis oculi muscle in the rat. *Journal of Neuroscience* 22:8808–18. [aBM]

Morin, A. (2001) The Split-Brain debate revisited: On the importance of language and self-recognition for right hemispheric consciousness. *Journal of Mind and Behavior* 22(2):107–18. [AM]

(2006) Levels of consciousness and self-awareness: A comparison and integration of various neurocognitive views. *Consciousness and Cognition* 15:358–71. [DC, CI, arBM]

Morris, D. (1967) *The naked ape: A zoologist's study of the human animal*. McGraw-Hill. [RBG]

Morris, J. S., DeGelder, B., Weiskrantz, L. & Dolan, R. J. (2001) Differential extrageniculostriate and amygdala responses to presentation of emotional faces in a cortically blind field. *Brain* 124(Pt 6):1241–52. [SW]

Morsella, E. (2005) The function of phenomenal states: Supramodular interaction theory. *Psychological Review* 112:1000–21. [EM]

Mort, E., Cairns, S., Hersch, H. & Finlay, B. (1980) The role of the superior colliculus in visually guided locomotion and visual orienting in the hamster. *Physiological Psychology* 8:20–28. [aBM]

Moruzzi, G. & Magoun, H. W. (1949) Brain stem reticular formation and activation of the EEG. *Electroencephalography and Clinical Neurophysiology* 1: 455–73. [arBM, DTW]

Moschovakis, A. K. (1996) Neural network simulations of the primate oculomotor system. II. Frames of reference. *Brain Research Bulletin* 40:337–45. [aBM]

Moschovakis, A. K. & Highstein, S. M. (1994) The anatomy and physiology of primate neurons the control rapid eye movements. *Annual Review of Neuroscience* 17:465–88. [aBM]

Mountcastle, V. B., ed. (1974) *Medical physiology*, 13th edition. C. V. Mosby. [WJF]

Mouton, L. J. (1999) *Spinal efferents and afferents of the periaqueductal gray: Possible role in pain, sex and micturition*. Doctoral dissertation, University of Groningen. [aBM]

Müller, J. R., Philiastides, M. G. & Newsome, W. T. (2004) Microstimulation of the superior colliculus focuses attention without moving the eyes. *Proceedings of the National Academy of Sciences USA* 102(30):524–29. [RJK, aBM]

Muller, U., von Cramon, D. Y. & Pollmann, S. (1998) D1- versus D2-receptor modulation of visuospatial working memory in humans. *Journal of Neuroscience* 18:2720–28. [FA]

The Multi-Society Task Force on the Persistent Vegetative State. (1994) Statement on medical aspects of the persistent vegetative state (first of two parts). *New England Journal of Medicine* 330:1499–1508. [aBM]

Mungamuru, S. S., Baldwin, B. A., Chindaduangratna C. & Kotchabhakdi, N. (2002) Hypothalamic and zona incerta neurons in sheep, initially only responding to the sight of food, also respond to the sight of water after intracerebroventricular injection of hypertonic saline or angiotensin II. *Brain Research* 925:204–12. [aBM]

Munoz, D. P. & Everling, S. (2004) Look away: The anti-saccade task and the voluntary control of eye movement. *Nature Reviews in Neuroscience* 5: 218–28. [FB]

Munoz, D. P. & Fecteau, J. H. (2002) Vying for dominance: Dynamic interactions control visual fixation and saccadic initiation in the superior colliculus. *Progress in Brain Research* 140:3–19. [RJK]

Myers, L. B. & Bulich. L. A. (2005) *Anesthesia for fetal intervention and surgery*. B. C. Decker. [RRB]

Myers, R. E. (1959) Cerebral ischemia in the developing primate fetus. *Biochimica Biochimica Acta* 48:S137–42. [aBM]

Nair, D. R., Najm, I., Bulacio J. & Luders. H. (2001) Painful auras in focal epilepsy. *Neurology* 57:700–702. [MD]

Nandi. D., Aziz, T., Carter, H. & Stein, J. (2003) Thalamic field potentials in chronic central pain treated by periventricular gray stimulation—a series of eight cases. *Pain* 101(1-2):97–107. [KJSA]

Narsinghani. U. & Anand, K. J. S. (2000) Developmental neurobiology of pain in neonatal rats. *Lab Animal* 29(9):27–39. [KJSA]

Nashold, B. S., Wilson, W. P. & Slaughter, G. (1969) Sensations evoked by stimulation of the midbrain of man. *Journal of Neurosurgery* 30:14–24. [aBM]

Neelon, M. F., Bringart, D. S. & Simpson, B. D. (2004) The isoazimuthal perception of sounds across distance: A preliminary investigation into the location of the audio egocenter. *Journal of Neuroscience* 24:7640–47. [aBM]

Nemec, P., Altman, J., Marhold, S., Burda, H. & Oelschläger, H. H. A. (2001) Neuroanatomy of magnetoreception: The superior colliculus involved in magnetic orientation in a mammal. *Science* 294:366–68. [aBM]

Newman, J. & Baars, B. J. (1993) A neural attentional model for access to consciousness: A Global Workspace perspective. *Concepts in Neuroscience* 4(2):255–90. [DFW]

Nicolelis, M. A. L., Chapin. J. K. & Lin. R. C. (1992) Somatotopic maps within the zona incerta relay parallel GABAergic somatosensory pathways to the neocortex, superior colliculus and brainstem. *Brain Research* 577:134–41. [aBM]

Niemi-Junkola, U. J. & Westby, C. W. M. (2000) Cerebellar output exerts spatially organised influence on neural responses in the rat superior colliculus. *Neuroscience* 97:565–73. [aBM]

Nieuwenhuis, R., Ten Donkelaar, H. J. & Nicholson, C., eds. (1998) *The central nervous system of vertebrates* (4 Vols.). Springer-Verlag. [aBM]

Northcutt, R. G. (1996a) The Agnathan ark: The origin of the craniate brain. *Brain, Behavior and Evolution* 48:237–47. [aBM]

(1996b) The origin of craniates: neural crest, neurogenic placodes, and homeobox genes. *Israel Journal of Zoology* 42:S273–13. [aBM]

Northcutt, R. G. & Wicht, H. (1997) Afferent and efferent connections of the lateral and medial pallia of the silver lamprey. *Brain, Behavior and Evolution* 49: 1–19. [aBM]

Northoff, G. & Bermpohl, F. (2004) Cortical midline structures and the self. *Trends in Cognitive Science* 8(5):102–107. [GN]

Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H. & Panksepp, j. (2006) Self-referential processing in our brain: A meta-analysis of imaging studies on the self. *Neuroimage* 31(1): 440–57. [aBM, GN]

Nudo, R. J. & Masterton, R. B. (1988) Descending pathways to the spinal cord: A comparative study of 22 mammals. *Journal of Comparative Neurology* 277: 53–79. [aBM]

Oakley, D. A. (1983) The varieties of memory: A phylogenetic approach. In: *Memory in animals and humans*. ed. A. Mayes, pp. 20–82. Van Nostrand Reinhold. [aBM]

Oakley, T. H. & Cunningham, C. W. (2002) Molecular phylogenetic evidence for the independent evolutionary origin of the arthropod compound eye. *Proceedings of the National Academy of Sciences USA* 99:1426. [DBE]

Ohman, A. (2005) The role of the amygdala in human fear: Automatic detection of threat. *Psychoneuroendocrinology* 30:953–58. [CI]

O'Keefe, J. & Nadel, L. (1978) *The hippocampus as a cognitive map*. Clarendon Press. [WJF]

O'Leary, D. D., Schlaggar. B. L. & Stanfield. B. B. (1992) The specification of sensory cortex: Lessons from cortical transplantation. *Experimental Neurology* 115(1):121–26. [KJSA]

Olson, E. J., Boeve. B. F. & Silber. M. H. (2000) Rapid eye movement sleep behaviour disorder: Demographic, clinical and laboratory findings in 93 cases. *Brain* 123:331–39. [DC]

Onofrj, M., Bonanni. L., Albani, G., Manzo, A., Bulla, D. & Thomas, A. (2006) Visual hallucinations in Parkinson's disease: Clues to separate origins *Journal of the Neurological Sciences* 248:143–50. [DC]

O'Regan, J. K., Deubel, H., Clark, J. J. & Rensink. R. A. (2000) Picture changes during blinks: Looking without seeing and seeing without looking. *Visual Cognition* 7:191–211. [aBM]

Orlovsky, G. N. & Shik, M. L. (1976) Control of locomotion: A neurophysiological analysis of the cat locomotor system. *International Review of Physiology* 10:281–317. [aBM]

O'Shea, R. P. & Corballis, P. M. (2005) Visual grouping on binocular rivalry in a split-brain observer. *Vision Research* 45:247–61. [EM]

Owen, A. M., Coleman, M. R., Boly, M., Davis, M. H., Laureys, S. & Pickard, J. D. (2006) Detecting awareness in the vegetative state. *Science* 313(5792):1402. [SW]

Özen, C., Augustine, C. J. & Hall. W. C. (2000) Contribution of superficial layer neurons to premotor bursts in the superior colliculus. *Journal of Neurophysiology* 84:460–71. [arBM]

Padel, Y. (1993) Magnocellular and parvocellular red nuclei. Anatomico-functional aspects and relations with the cerebellum and other nerve centres. [Article in French]. *Revue Neurologique (Paris)* 149:703–15. [aBM]

Palmer, L. A., Rosenquist, A. C. & Sprague, J. M. (1972) Corticotectal systems in the cat: Their structure and function. In: *Corticothalamic projections and sensorimotor activities*, ed. T. Frigyesi, E. Rinvik & M. D. Yahr, pp. 491–522. Raven Press. [aBM]

Panksepp, J. (1982) Toward a general psychobiological theory of emotions. *Behavioral and Brain Sciences* 5:407–67. [arBM]

(1998a) *Affective neuroscience. The foundations of human and animal emotions.* Oxford University Press. [CI, aBM, GN, JF, DFW]

(1998b) The periconscious substrates of consciousness: Affective states and the evolutionary origins of the self. *Journal of Consciousness Studies* 5(5–6): 566–82. [GN, JP]

(2003) At the interface of the affective, behavioral, and cognitive neurosciences: Decoding the emotional feelings of the brain. *Brain and Cognition* 52(1): 4–14. [GN]

(2005a) Affective consciousness: Core emotional feelings in animals and humans. *Consciousness and Cognition* 14(1):30–80. [GN, JP, JvH]

(2005b) On the embodied neural nature of core emotional affects. *Journal of Consciousness Studies* 12:158–84. [CI]

(2006) Emotional endophenotypes in evolutionary psychiatry. *Progress in Neuro-Psychopharmacology and Biological Psychiatry* 30:774–84. [JP]

Panksepp, J. & Burgdorf, J. (1999) Laughing rats? Playful tickling arouses high frequency ultrasonic chirping in young rodents. In: *Toward a science of consciousness, vol. III*, ed. S. Hameroff, D. Chalmers & A. Kazniak, pp. 231–44. MIT Press. [JP]

(2003) "Laughing" rats and the evolutionary antecedents of human joy? *Physiology and Behavior* 79:533–47. [JP]

Panksepp, J., Normansell, L., Cox, J. F. & Siviy, S. M. (1994) Effects of neonatal decortication on the social play of juvenile rats. *Physiology and Behavior* 56:429–43. [aBM, JP]

Panksepp, J. & Panksepp, J. B. (2000) The seven sins of evolutionary psychology. *Evolution and Cognition* 6:108–31. [JvH]

Parmelee, P. A. (1996) Pain in cognitively impaired older persons. *Clinics in Geriatric Medicine* 12(3):473–87. [KJSA]

Parvizi, J. & Damasio, A. R. (2001) Consciousness and the brainstem. *Cognition* 79:135–60. [aBM]

(2003) Neuroanatomical correlates of brainstem coma. *Brain* 126:1524–36. [aBM]

Pellis, S. M., Pellis, V. C. & Whishaw, I. Q. (1992) The role of the cortex in play fighting by rats: Developmental and evolutionary implications. *Brain, Behavior and Evolution* 39:270–84. [aBM]

Penfield, W. (1952) Epileptic automatisms and the centrencephalic integrating system. *Research Publications of the Association for Nervous and Mental Disease* 30:513–28. [FA, aBM]

Penfield, W. & Jasper, H. H. (1954) *Epilepsy and the functional anatomy of the human brain.* Little, Brown. [KJSA, R-PB, RRE, AMLC, DBE, WJF, RBC, aBM]

Penfield, W. & Rasmussen, T. (1955) *The cerebral cortex of man.* MacMillan. [MD]

Pereira, L. C., Modesto, A. M., Sugai, R. & daMota, L. A. (2005) Pain sensitive cerebral areas and intracranial structures revealed at fully awake craniotomies for primary intracranial tumor resection. *Abstracts IASP 11th World Congress on Pain*, 1547:P20. IASP Press. [MD]

Pernick, M. S. (1985) "They don't feel it like we do": Social politics and the perception of pain. In: *A calculus of suffering. Pain, professionalism, and anesthesia in nineteenth-century America*, by M. S. Pernick, pp. 148–70. Columbia University Press. [KJSA]

Perry, D. C. & Kellar, K. J. (1995) [3H]epibatidine labels nicotinic receptors in rat brain: An autoradiographic study. *Journal of Pharmacology and Experimental Therapeutics* 275:1030–34. [DC]

Perry, E. K., Court, J. A., Johnson, M., Smith, C. J., James, V., Cheng, A. V., Kerwin, J. M., Morris, C. M., Piggott, M. A., Edwardson, J. A., Birdsall, N. J. M., Turner, J. T. & Perry, R. H. (1993) Autoradiographic comparison of cholinergic and other transmitter receptors in the normal human hippocampus. *Hippocampus* 3:307–15. [DC]

Perry, E. K., Irving, D., Kerwin, J. M., McKeith, I. G., Thompson, P., Collerton, D., Fairbairn, A. F., Ince, P. G., Morris, C. M., Cheng, A. V. & Perry, R. H. (1993) Cholinergic transmitter and neurotrophic activities in Lewy body dementia: Similarity to Parkinson's and distinction from Alzheimer disease. *Alzheimer Disease and Associated Disorders* 7:69–79. [DC]

Perry, E. K., Morris, C. M., Court, J. A., Cheng, A., Fairbairn, A. F., McKeith, I. G., Irving, D., Brown, A. & Perry, R. H. (1995) Alteration in nicotine binding sites in Parkinson's disease, Lewy body dementia and Alzheimer's disease: Possible index of early neuropathology. *Neuroscience* 64:385–95. [DC]

Perry, E. K. & Piggott, M. A. (2000) Neurotransmitter mechanisms of dreaming: Implications of modulatory systems abased on dream intensity. *Behavioral and Brain Sciences* 23:990–92. [DC]

Perry, E. K., Walker, M., Grace, J. & Perry, R. (1999) Acetylcholine in mind: A neurotransmitter correlate of consciousness? *Trends in the Neurosciences.* 22:273–80. [DC]

Persaud, N., McLeod, P. & Cowey, A. (2007) Post-decision wagering objectively measures awareness. *Nature Neuroscience* 10:257–61. [rBM]

Petrides, M. & Pandya, D. N. (2002) Association pathways of the prefrontal cortex and functional observations. In: *Principles of frontal lobe function*, ed. D. T. Stuss & R. T. Knight, pp. 31–50. Oxford University Press. [FB]

Petty, F. G. (1998) Consciousness: A neurosurgical perspective. *Journal of Consciousness Studies* 5:86–95. [aBM]

Peyron, R., Laurent, B. & Garcia-Larrea, L. (2000) Functional imaging of brain responses to pain: A review and meta-analysis. *Neurophysiologie Clinique* 30:263–88. [MD]

Phan, K. L., Taylor, S. F., Welsh, R. C., Ho, S. H., Britton, J. C. & Liberzon, I. (2004) Neural correlates of individual ratings of emotional salience: A trial-related fMRI study. *Neuroimage* 21(2): 768–80. [GN]

Philipona, D., O'Reagan, J. K. & Nadal, J.-P. (2003) Is there something out there? Inferring space from sensorimotor dependencies. *Neural Computation* 15:2029–43. [aBM]

Philipona, D., O Regan, J. K., Nadal, J.-P. & Coenen, O. J.-M. D. (2004) Perception of the structure of the physical world using multimodal unknown sensors and effectors. *Advances in Neural Information Processing Systems* 16: 945–52. [aBM]

Phillips, P. E., Stuber, G. D., Heien, M. L., Wightman, R. M. & Carelli, R. M. (2003) Subsecond dopamine release promotes cocaine seeking. *Nature* 422:614–18. [FA]

Pierrot-Deseilligny, C., Rivaud, S., Gaymard, B. & Agid, Y. (1991) Cortical control of reflexive visually-guided saccades. *Brain* 114:1473–85. [FB]

Pinkott, S. L., Piggott, M., Ballard, C., McKeith, I., Perry, R., Konieta, S., Owens, J., Wyper, D. & Perry, E. (2006) Thalamic nicotinic receptors implicated in disturbed consciousness in dementia with Lewy bodies. *Neurobiology of Disease* 21:50–56. [DC]

Plotnik, J. M., de Waal, F. B. M. & Reiss, D. (2006) Self-recognition in an Asian elephant. *Proceedings of the National Academy of Sciences USA* 103: 17053–17057. [EG]

Pockett, S. (2006) The great subjective back-referral debate: Do neural responses increase during a train of stimuli? *Consciousness and Cognition* 15:551–59. [rBM]

Pollen, D. A. (2003) Explicit neural representations, recursive neural networks and conscious visual perception. *Cerebral Cortex* 13:807–14. [rBM]

Pombal, M. A., El Manira, A. & Grillner, S. (1997) Afferents of the lamprey striatum with special reference to the dopaminergic system: A combined tracing and immunohistochemical study. *Journal of Comparative Neurology* 386:71–91. [rBM]

Pöppel, E. & Richards, W. (1974) Light sensitivity in cortical scotomata contralateral to small islands of blindness. *Experimental Brain Research* 21:125–30. [rBM]

Populin, L. C. & Yin, T. C. T. (1998) Sensitivity of auditory cells in the superior colliculus to eye position in the behaving cat. In: *Psychological and physiological advances in hearing*, ed. A. R. Palmer, A. Q. Summerfield & R. Meddis, pp 441–48. Whurr. [aBM]

Porges, S. W. (2001) The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology* 42:123–46. [JvH]

Porter, F. L. & Anand, K. J. S. (1998) Epidemiology of pain in neonates. *Research & Clinical Forums* 20(4):9–16. [KJSA]

Posner, M. I. & Petersen, S. E. (1990) The attention systems of the human brain. *Annual Review of Neuroscience* 13:25–42. [FB, aBM]

Posner, M. I. & Rothbart, M. (2000) Developing mechanisms of self-regulation. *Development and Psychopathology* 12:427–41. [CI]

Povinelli, D. J. (1989) Failure to find self-recognition in Asian elephants (*Elephas maximus*) in contrast to their use of mirrors cues to discover hidden food. *Journal of Comparative Psychology* 102:122–31. [EG]

Power, B. D., Kolmac, C. I. & Mitrofanis, J. (1999) Evidence for a large projection from the zona incerta to the dorsal thalamus. *Journal of Comparative Neurology* 404:554–65. [aBM]

Power, B. D. & Mitrofanis, J. (1999) Evidence for extensive inter-connections within the zona incerta in rats. *Neuroscience Letters* 267:9–12. [aBM]

(2001) Zona incerta: Substrate for the contralateral interconnectivity in the thalamus of rats. *Journal of Comparative Neurology* 436:52–63. [aBM]

Prescott, T. J., Redgrave, P. & Gurney, K. N. (1999) Layered control architectures in robots and vertebrates. *Adaptive Behavior* 7(1):99–127. [arBM, TJP, AKS]

Provine, R. R. & Yong, Y. L. (1991) Laughter: A stereotyped human vocalization. *Ethology* 89:115–24. [aBM]

Quinn, P. C., Slater, A. M., Brown, E. & Hayes, R. A. (2001). Developmental change in form categorization in early infancy. *British Journal of Developmental Psychology* 19:207–18. [CI]

Rada, E. M. Tharakan, E. C. & Flood, P. (2003) Volatile anesthetics reduce agonist affinity at nicotinic acetylcholine receptors in the brain. *Anesthesia and Analgesia* 96:108–11. [DC]

Rainville, P., Duncan, G. H., Price, D. D., Carrier, B. & Bushnell, M. C. (1997) Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science* 277:968–71. [MD]

Ramón-Moliner, E. & Nauta, W. J. H. (1966) The isodendritic core of the brainstem. *Journal of Comparative Neurology* 126:311–35. [aBM]

Rankin, C. H. (2002) From gene to identified neuron to behaviour in *Caenorhabditis elegans*. *Nature Reviews Genetics* 3:622–30. [SMD]

Ray, M., Rohr, U., McIntosh, J. M., Ballard, C., McKeith, I., Chalon, S., Guilloteau, D., Perry, R., Perry, E., Court, J. A. & Piggott, M. (2004) Involvement of alpha6/alpha3 neuronal nicotinic acetylcholine receptors in neuropsychiatric features of Dementia with Lewy bodies: (123)I-alpha-conotoxin MII binding in the thalamus and striatum. *Neuroscience Letters* 372:220–25. [DC]

Raz, A. & Buhle, J. (2006) Typologies of attentional networks. *Nature Reviews Neuroscience* 7:367–79. [aBM]

Reddy, L., Reddy, L. & Koch, C. (2006) Face identification in the near-absence of focal attention. *Vision Research* 46:2336–2343. [rBM]

Redgrave, P. & Gurney, K. (2006) The short-latency dopamine signal: A role in discovering novel actions? *Nature Reviews Neuroscience* 7:967–75. [PA]

Redgrave P., McHaffie, J. C. & Stein, B. E. (1986a) Nociceptive neurons in rat superior colliculus I: Antidromic activation from the contralateral predorsal bundle. *Experimental Brain Research* 109:185–96. [rBM]

Redgrave, P., Prescott, T. & Gurney, K. N. (1999) The basal ganglia: A vertebrate solution to the selection problem? *Neuroscience* 89:1009–23. [TJP]

Redgrave, P., Simkins, M., McHaffie, J. G. & Stein, B. E. (1986b) Nociceptive neurons in rat superior colliculus II: Effects of lesions to contralateral descending output pathway on nociffensive behaviors. *Experimental Brain Research* 109:197–208. [rBM]

Rees, G. (2001) Neuroimaging of visual awareness in patients and normal subjects. *Current Opinion in Neurobiology* 11:150–56. [aBM]

Rees, G., Kreiman, G. & Koch, C. (2002) Neural correlates of consciousness in humans. *Nature Reviews Neuroscience* 3(4):261–70. [SMD, SW]

Rees, G. & Lavie, N. (2001) What can functional imaging reveal about the role of attention in visual awareness? *Neuropsychologia* 39:1343–53. [aBM]

Reiner, A., Yamamoto, K. & Karten, H. J. (2005) Organization and evolution of the avian forebrain. *Anatomical Record Part A, Discoveries in Molecular, Cellular, and Evolutionary Biology* 287(1):1080–102. [DBE]

Reiss, D. & Marino, L. (2001) Mirror self-recognition in the bottlenose dolphin: A case of cognitive convergence. *Proceedings of the National Academy of Sciences USA* 98:5937–42. [EG]

Rensink, R.A. (2002) Change detection. *Annual Review of Psychology* 53: 245–77. [aBM]

Rensink, R.A., O'Regan, J. K. and Clark, J. J. (1997) To see or not to see: The need for attention to perceive changes in scenes. *Psychological Science* 8: 368–73. [aBM]

Ressler, N. (2004) Rewards and punishments, goal-directed behavior and consciousness. *Neuroscience and Biobehavioral Reviews* 28:27–39. [JvH]

Richards, W., Seung H. S. & Pickard, C. (2006) Neural voting machines. *Neural Networks* 19:1161–67. [aBM]

Rieck, R. W., Huerta, M. F., Harting, J. K. & Weber, J. T. (1986) Hypothalamic and ventral thalamic projections to the superior colliculus in the cat. *Journal of Comparative Neurology* 243:249–65. [aBM]

Rock, I. (1995) *Perception*. Scientific American Library/W. H. Freeman. [RBG]

Rockoß, C. O. (1959) Considerations on the visual egocenter. *Acta Psychologica* 16:226–34. [aBM]

Rolls, E. T. (1999) *The brain and emotion*. Oxford University Press. [JvH]

Rosenthal, D. M. (2002) How many kinds of consciousness? *Consciousness and Cognition* 11:653–65. [CI]

Rosseti, Z. L. & Carboni, S. (2005) Noradrenaline and dopamine elevations in the rat prefrontal cortex in spatial working memory. *Journal of Neuroscience* 25(9):2322–29. [PA]

Roth, G. (1987) *Visual behavior in salamanders*. Springer-Verlag. [WJH]

Royal College of Obstetricians and Gynecologists (1997) *Fetal Awareness: Report of a Working Party*. RCOG Press. [RRB]

Rushmore, R. J., Valero-Cabre A., Lomber, S. G., Hilgetag, C. C. & Payne, B. R. (2005) Functional circuitry underlying visual neglect. *Brain* 29:1963–21. [rBM]

Sachs, E. (1967) Dissociation of learning in rats and its similarities to dissociative states in man. In: *Comparative psychopathology: Animal and human*, ed. J. Zubin & H. Hunt, pp. 249–304. Grune & Stratton. [rBM]

Sahraie, A., Weiskrantz, L., Barbur, J. L. Simmons, A., Williams, S. C. & Brammer, M. J. (1997) Pattern of neuronal activity associated with conscious and unconscious processing of visual signals. *Proceedings of the National Academy of Sciences USA* 94(17):9406–11. [SW]

Sakuma, Y. & Pfaff, D. W. (1979) Facilitation of female reproductive behavior from mesencephalic central gray in the rat. *American Journal of Physiology* 237:R278–84. [aBM]

Satterlie, R. A. & Nolen, T. G. (2001) Why do cubomedusae have only four swim pacemakers? *Journal of Experimental Biology* 204:1413–19. [aBM]

Schaefer, K. P. & Schneider, H. (1968) Reizversuche im tectum opticum des Kaninchens. Ein experimenteller Beitrag zur sensorimotorischen Koordination des Hirnstammes. *Archiv für Psychiatrie und Nervenkrankheiten* 211:118–37. [aBM]

Schall, J. D. (1997) Visuomotor areas of the frontal lobe. *Cerebral Cortex* 12: 527–638. [aBM]

Schaller, G. B. (1972) *The Serengeti lion*. University of Chicago Press. [EG]

Scheibel, M. E. & Scheibel, A. B. (1967) Anatomical basis of attention mechanisms in vertebrate brains. In: *The neurosciences, a study program*, ed. G. C. Quarton, T. Melnechuk & F. O. Schmitt, pp. 577–602. Rockefeller University Press. [TJP]

(1977) The anatomy of constancy. *Annals of the New York Academy of Sciences* 290:421–35. [aBM]

Schiller, P. H. & Koerner, F. (1971) Discharge characteristics of single units in superior colliculus of the alert Rhesus monkey. *Journal of Neurophysiology* 43:930–36. [aBM]

Schiller, P. H. & Lee, K. (1994) The effects of lateral geniculate nucleus, area V4, and middle temporal (MT) lesions on visually guided eye movements. *Visual Neuroscience* 11:229–41. [aBM]

Schiller, P. H., True, S. D. & Conway, J. L. (1979) Effects of frontal eye field and superior colliculus ablations on eye movements. *Science* 206:590–92. [aBM]

Schlag-Rey, M., Schlag, J. & Dassonville, P. (1992) How the frontal eye fields impose a saccade goal on superior colliculus neurons. *Journal of Neurophysiology* 67:1003–1005. [aBM]

Schmidtke, K. & Buttner-Ennever, J. A. (1992) Nervous control of eyelid function. A review of clinical, experimental and pathological data. *Brain* 115:227–47. [aBM]

Schneider, F., Rotte, M., Dahlem, Y. Bogerts, B., Tempelmann, C., Heinze, H. J. Walter, M. & Northoff, G. (submitted) Modulation of the resting state in cortical midline structures by self-relatedness. [GN]

Schneider, G. E. (1967) Contrasting visuomotor functions of tectum and cortex in the golden hamster. *Psychologische Forschung* 31:52–62. [aBM]

Schneider, K. A. & Kastner, S. (2005) Visual responses of the human superior colliculus: A high-resolution functional magnetic resonance imaging study. *Journal of Neurophysiology* 94(4):2491–03. [SW]

Schneider, W. X. & Deubel, H. (2002) Selection-for-perception and selection-for-spatial-motor-action are coupled by visual attention: A review of recent findings and new evidence from stimulus-driven saccade control. In: *Attention and performance XIX: Common mechanisms in perception and action*, ed. W. Prinz & B. Hommel, pp. 609–27. Oxford University Press. [aBM]

Schooler, J. W. (2002) Re-representing consciousness: Dissociations between experience and meta-consciousness. *Trends in Cognitive Sciences* 6(8): 339–44. [AM]

Schopenhauer, A. (1819/1958) *The world as will and representation* (2 vols.), trans. E. F. J. Payne. Dover. [aBM, R-PB]

Schuller, G. & Radtke-Schuller, S. (1990) Neural control of vocalization in bats: Mapping of brainstem areas with electrical microstimulation eliciting species-specific echolocation calls in the rufous horseshoe bat. *Experimental Brain Research* 79:192–206. [aBM]

Schultz, W. & Dickinson, A. (2000) Neuronal coding of prediction errors. *Annual Review of Neuroscience* 23:473–500. [PA]

Schutter, D. J. L. G. & Van Honk, J. (2004a) Extending the global workspace theory to emotion: Phenomenality without access. *Consciousness and Cognition* 13:539–49. [JvH]

(2004b) Schizophrenia: A disorder of affective consciousness. *Behavioral and Brain Sciences* 27:804–805. [JvH]

Schwartz, B. L. (2005) Do nonhuman primates have episodic memory? In: *The missing link in cognition. Origins of self-reflective consciousness*, ed. H. S. Terrace & J. Metcalfe, pp. 225–41. Oxford University Press. [AM]

Searle, J. R. (1983) *Intentionality: An essay on the philosophy of mind*. Cambridge University Press. [JP]

(1992) *The rediscovery of the mind*. MIT Press/Bradford Books. [R-PB, aBM]

(1997) *The mystery of consciousness*. Granta. [R-PB]

Seth, A. K. (2005) Causal connectivity analysis of evolved neural networks during behavior. *Network: Computation in Neural Systems* 16(1):35–55. [AKS]

(in press) The ecology of action selection: Insights from artificial life. *Philosophical Transactions of the Royal Society of London, B: Biological Sciences*. [AKS]

Seth, A. K., Baars, B. J. & Edelman, D. B. (2005) Criteria for consciousness in humans and other mammals. *Consciousness and Cognition* 14(1):119–39. [DBE, rBM, AKS]

Seth, A. K., Izhikevich. E., Reeke, G. N. & Edelman. G. M. (2006) Theories and measures of consciousness: An extended framework. *Proceedings of the National Academy of Sciences USA* 103(28):10799–804. [AKS]

Sewards, T. V. & Sewards, M. A. (2000) Visual awareness due to neuronal activities in subcortical structures: A proposal. *Consciousness and Cognition* 9: 86–116. [arBM]

Shapiro, B. S. (1999) Implications for our definitions of pain. *Pain Forum* 8: 100–102. [KJSA]

Shapiro, L. A. (2004) *The mind incarnate*. MIT Press. [GP]

Sharp, P. E., Blair, H. T. & Cho, J. (2001) The anatomical and computational basis of the rat head-direction cell signal. *Trends in Neurosciences* 24:289–94. [rBM]

Shepherd, G. M. & Greer, C. A. (1998) Olfactory bulb. In: *The synaptic organization of the brain*. 4th edition, ed. G. M. Shepherd, pp. 159–204. Oxford University Press. [RM]

Shergill, S. S., Brammer, M. J., Williams, S. C., Murray, R. M. & McGuire, P. K. (2000) Mapping auditory hallucinations in schizophrenia using functional magnetic resonance imaging. *Archives of General Psychiatry* 57: 1033–38. [R-PH]

Sherman, H. B., Caviness, V.S., Jr. & Ingle, D. J. (1979) Corticotectal connections in the gerbil. *Neuroscience Abstract* 9:120. [aBM]

Sherman, S. M. (1974) Visual fields of cats with cortical and tectal lesions. *Science* 185:355–57. [aBM]

(1977) The effect of superior colliculus lesions upon the visual fields of cats with cortical ablations. *Journal of Comparative Neurology* 172:211–30. [aBM]

Sherman, S. M. & Guillery, R. W. (2001) *Exploring the thalamus*. Academic Press. [aBM]

Shewmon, D. A. (2004) The ABC of PVS. In: *Brain death and disorders of consciousness*, ed. C. Machado & D. A. Shewmon, pp. 515–28. Kluwer Academic/ Plenum. [aBM]

Shewmon, D. A., Capron, A. M., Peacock, W. J. & Shulman, B. L. (1989) The use of anencephalic infants as organ sources. A critique. *Journal of the American Medical Association* 261:1773–81. [aBM]

Shewmon, D. A., Holmes, G. L. & Byrne. P. A. (1999) Consciousness in congenitally decorticate children: Developmental vegetative state as self-fulfilling prophecy. *Developmental Medicine and Child Neurology* 41:364–74. [DBE, aBM, JP]

Shieber, S. M., ed. (2004) *The Turing test: Verbal behavior as the hallmark of intelligence*. MIT Press. [RBG]

Shiller, V. M., Izard, C. E. & Hembree, E. A. (1986) Patterns of emotion expression during separation in the Strange Situation procedure. *Developmental Psychology* 22:378–82. [CI]

Silvia, P. J. (2006) *Exploring the psychology of interest*. Oxford University Press. [CI]

Simons, D. J. & Chabris, C. F. (1999) Gorillas in our midst: Sustained inattentional blindness for dynamic events. *Perception* 28:1059–74. [aBM]

Simons, S. H. P., van Dijk, M., Anand, K. J. S. Roofthooft, D., van Lingen, R. A. & Tibboel, D. (2003) Do we still hurt newborn babies? A prospective study of procedural pain and analgesia in neonates. *Archives of Pediatrics and Adolescent Medicine* 157:1058–64. [KJSA]

Singer, W. (2001) Consciousness and the binding problem. In: *Cajal and consciousness: Scientific approaches to consciousness on the centennial of Ramón y Cajal's Textura*, ed. P. C. Marijuán. *Annals of the New York Academy of Science* 929:123–46. [aBM]

Slater, R., Cantarella. A., Gallella, S., Worley, A., Boyd, S., Meek, J. & Fitzgerald, M. (2006) Cortical pain responses in human infants. *Journal of Neuroscience* 26(14):3662–66. [KJSA]

Smeets, W. J. A. J., Marín, O. & González, A. (2000) Evolution of the basal ganglia: New perspectives through a comparative approach. *Journal of Anatomy* 196:501–17. [rBM]

Smith, S. (1966) *Commission of inquiry into fetal sentience*. CARE. [RRB]

Snaith, S. & Holland, O. (1990) An investigation of two mediation strategies suitable for behavioural control in animals and animats. In: *From animals to animats: Proceedings of the First International Conference on Simulation of Adaptive Behavior*, ed. J.-A. Meyer & S. Wilson, pp. 255–62. MIT Press. [aBM]

Snodgrass, R. E. (1935) *Principles of insect morphology*. McGraw-Hill. [aBM]

Sokolov, E. N. (1963) *Perception and the conditioned reflex*. Pergamon Press. [FH]

Sommer, M. A. & Wurtz, R. H. (2004) What the brain stem tells the frontal cortex. I. Oculomotor signals sent from superior colliculus to frontal eye field via mediodorsal thalamus. *Journal of Neurophysiology* 91:1381–402. [aBM]

Sparks, D. L. (1999) Conceptual issues related to the role of the superior colliculus in the control of gaze. *Current Opinion in Neurobiology* 9:698–707. [aBM]

Sparks, D. L. & Jay, M. F. (1986) The functional organization of the primate superior colliculus: A motor perspective. *Progress in Brain Research* 64: 235–241. [FA]

Sperry. R. W. (1952) Neurology and the mind-brain problem. *American Scientist*, 40:291–312. [EM]

Sporns, O., Tononi, G. & Edelman. G. M. (2000) Theoretical neuroanatomy: Relating anatomical and functional connectivity in graphs and cortical connection matrices. *Cerebral Cortex* 10:127–41. [aBM, AKS]

Sprague, J. M. (1966) Interaction of cortex and superior colliculus in mediation of visually guided behavior in the cat. *Science* 153:1544–47. [aBM]

(1975) Mammalian tectum: Intrinsic organization, afferent inputs and integrative mechanisms. In: *Sensorimotor function of the midbrain tectum*, ed. D. Ingle & J. M. Sprague. *Neurosciences Research Program Bulletin* 13:204–14. MIT Press. [aBM]

(1991) The role of the superior colliculus in facilitating visual attention and form perception. *Proceedings of the National Academy of Sciences USA* 88: 1286–90. [aBM]

(1996) Neural mechanisms of the visual orienting response. *Progress in Brain Research* 112:1–15. [aBM, SW]

Sprague, J. M., Chambers, W. W. & Stellar, E. (1961) Attentive, affective, and adaptive behavior in the cat. *Science* 133:165–73. [aBM]

Spurden, D. P., Court, J. A., Lloyd, S., Oakley, A., Perry, R., Pearson, C., Pullen, R. G. & Perry, E. K. (1997) Nicotinic receptor distribution in the human thalamus: Autoradiographical localization of ^{3}H]nicotine and [^{125}I] alpha-bungarotoxin binding. *Journal of Chemical Neuroanatomy* 13:105–13. [DC]

Stallard, P., Williams, L., Lenton, S. & Velleman, R. (2001) Pain in cognitively impaired, non-communicating children. *Archives of Disease in Childhood* 85(6):460–462. [KJSA]

Standing, L. (1973) Learning 10,000 pictures. *Quarterly Journal of Experimental Psychology* 25:207–22. [aBM]

Steele, G. E. & Weller. R. E. (1993) Subcortical connections of subdivisions of inferior temporal cortex in squirrel monkeys. *Visual Neuroscience* 10: 563–83. [aBM]

Stefan, H. & Snead, O. C. (1997) Absence seizures. In *Epilepsy: A comprehensive textbook*, ed. J. Engel, Jr. & T. A. Pedley, pp. 579–90. Lippincott-Raven. [aBM]

Steriade, M. (2001) Impact of network activities on neuronal properties in corticothalamic systems. *Journal of Neurophysiology* 86:1–39. [aBM]

Steriade, M., Jones, E. & Llinas, R. (1990) *Thalamic oscillations and signaling*. Wiley. [SMD]

Stoerig, P. & Barth, E. (2001) Low-level phenomenal vision despite unilateral destruction of primary visual cortex. *Consciousness and Cognition* 10: 574–87. [arBM]

Strafstrom, C. E. (2006) Epilepsy: A review of selected clinical syndromes and advances in basic science. *Journal of Cerebral Blood Flow and Metabolism* 26:983–1004. [aBM]

Strigo, I. A., Duncan, G. H., Boivin, M. & Bushnell, M. C. (2003) Differentiation of visceral and cutaneous pain in the human brain. *Journal of Neurophysiology* 89:3294–303. [MD]

Stuphorn, V., Bauswein, E. & Hoffmann, K.-P. (2000) Neurons in the primate superior colliculus coding for arm movements in gaze-related coordinates. *Journal of Neurophysiology* 83:1283–98. [aBM]

Summer, P., Tsai, F. C., Yu, E. & Nachev, P. (2006) Attentional modulation of sensorimotor processes in the absence of perceptual awareness. *Proceedings of the National Academy of Sciences USA* 103(27):10520–25. [SW]

Sutton. L. N., Bruce, D. A. & Schut. L. (1980) Hydranencephaly versus maximal hydrocephalus: An important clinical distinction. *Neurosurgery* 6: 34–38. [aBM]

Swanson, L. W. (1987) The hypothalamus. In: *Handbook of chemical neuroanatomy. Integrated systems of the CNS. Part I*, vol. 5, ed. T. Hökfelt, A. Björklund & L. W. Swanson, pp. 1–124. Elsevier. [aBM]

(2000) Cerebral hemisphere regulation of motivated behavior. *Brain Research* 886:113–64. [arBM]

Sylvester, R., Driver, J. & Rees, G. (2007) Visual fMRI responses in human superior colliculus show a temporal-nasal asymmetry that is absent in lateral geniculate and visual cortex. *Journal of Neurophysiology* 86:1405–502. [SW]

Takada, K., Shioka, M., Ando, M., Kimura, M. & Inone, K. (1989) Porencephaly and hydranencephaly: a neuropathological study of four autopsy cases. *Brain and Development* 11:51–56. [aBM]

Tarsitano, M. S. & Andrew, R. (1999) Scanning and route selection in the jumping spider *Portia labiata*. *Animal Behaviour* 58:255–65. [RG]

Tehovnik, E. J., Lee, K. & Schiller, P. H. (1984) Stimulation-evoked saccades from the dorsomedial frontal cortex of the rhesus monkey following lesions of the frontal eye fields and superior colliculus. *Experimental Brain Research* 98:179–90. [aBM]

Telford, S., Wang, S. & Redgrave, P. (1996) Analysis of nociceptive neurones in the rat superior colliculus using c-fos immunohistochemistry. *Journal of Comparative Neurology* 375:601–17. [rBM]

ten Donkelaar, H. J. (1988) Evolution of the red nucleus and rubrospinal tract. *Behavioral Brain Research* 28:920. [aBM]

Termine, N. T. & Izard, C. E. (1988) Infants' responses to their mothers' expressions of joy and sadness. *Developmental Psychology* 24:223–29. [CI]

Terrace, H. S. & Metcalfe, J., eds. (2005) *The missing link in cognition: Origins of self-reflective consciousness*. Oxford University Press. [AM]

Thompson, R. (1993) Centrencephalic theory, the general learning system, and subcortical dementia. *Annals of the New York Academy of Sciences* 702: 197–223. [FA, aBM, TJP]

Thompson, R. & Bachman, M. K. (1979) Zona incerta: A link between the visual cortical sensory system and the brainstem motor system. *Physiological Psychology* 7:251–53. [aBM]

Tinbergen, N. (1951) *The study of instinct*. Clarendon Press. [aBM]

Tirabuschi, P., Hansen, L. A., Alford, M., Merdes, A., Masliah, E., Thal, L. J. & Corey-Bloom, J. (2002) Early and widespread cholinergic losses differentiate dementia with Lewy bodies from Alzheimer disease. *Archives of General Psychiatry* 59:946–51. [DC]

Tobler, P. N., Dickinson, A. & Schultz, W. (2003) Coding of predicted reward omission by dopamine neurons in a conditioned inhibition paradigm. *Journal of Neuroscience* 23:10402–10. [FA]

Todd, P. M. & Gigerenzer, G. (2000). Précis of *Simple heuristics that make us smart*. *Behavioral and Brain Sciences* 23:727–81. [RAG]

Tong, F. (2003) Primary visual cortex and visual awareness. *Nature Reviews Neuroscience* 4:219–20. [rBM]

Tononi, G. (2004) An information integration theory of consciousness. *BMC Neuroscience* 5(1):42. [SMD, AKS]

Tononi, G. & Edelman, G. M. (1998) Consciousness and complexity. *Science* 282(5395):1846–51. [SMD, AKS]

Trageser, J. C., Burke, K. A., Masri, R., Li, Y., Sellers, L. & Keller, A. (2006) State-dependent gating of sensory inputs by zona incerta. *Journal of Neurophysiology* 96:1456–63. [TJP]

Trageser, J. C. & Keller, A. (2004) Reducing the uncertainty: Gating of peripheral inputs by zona incerta. *Journal of Neuroscience* 24:8911–9005. [aBM]

Trzch, D. D., Farrol, H. S., Duthie, E. H., Goldstein, M. D. & Lane, P. S. (1991) Clinical characteristics of patients in the persistent vegetative state. *Archives of Internal Medicine* 151:930–32. [rBM]

Trevarthen, C. & Reddy, V. (2006) Consciousness in infants. In: *The Blackwell companion to consciousness*, ed. M. Velmans & S. Schneider, pp. 41–57. Blackwell. [aBM]

Tronick, E. Z. & Cohn, J. F. (1989) Infant-mother face-to-face interaction: Age and gender differences in coordination and the occurrence of miscoordination. *Child Development* 60:85–92. [CI]

Tyrrell, T. (1993) *Computational mechanisms for action selection*. Doctoral dissertation. Centre for Cognitive Science, University of Edinburgh. [aBM]

Ugolini, G. (1995) The specificity of rabies virus as a transneuronal tracer of motor networks: Transfer from hypoglossal motoneurons to connected second-order and higher order central nervous system cell groups. *Journal of Comparative Neurology* 356:457–80. [aBM]

Uflig, N., Neudorfer, F. & Böhl, J. (2000) Transient structures of the human fetal brain: Subplate, thalamic reticular complex, ganglionic eminence. *Histology and Histopathology* 15(3):771–90. [KJSA]

Ungless, M. A., Magill, P. J. & Bolam, J. P. (2004) Uniform inhibition of dopamine neurons in the ventral tegmental area by aversive stimuli. *Science* 303: 2040–42. [FA]

Valentine, D. E. & Moss, C. F. (1997) Spatially selective auditory responses in the superior colliculus of the echolocating bat. *Journal of Neuroscience* 17: 1720–33. [aBM]

Vanderwolf, C. H., Kolb, B. & Cooley, R. K. (1978) Behavior of the rat after removal of the neocortex and hippocampal formation. *Journal of Comparative and Physiological Psychology* 92:156–75. [aBM]

van Hooff, J. A. R. A. M. (1972) A comparative approach to the phylogeny of laughter and smile. In: *Nonverbal communication*, ed. R. A. Hinde, pp. 209–41. Cambridge University Press. [aBM]

Van Honk, J. & Schutter, D. J. L. G. (2005) Dynamic brain systems in quest for emotional homeostasis. *Behavioral and Brain Sciences* 28:220–21. [JvH]

Van Opstal, A. J., Hepp, K., Suzuki, Y. & Henn, V. (1995) Influence of eye position on activity in monkey superior colliculus. *Journal of Neurophysiology* 74:1593–1610. [aBM]

van Rlezen, H. & Leonard, B. E. (1990) Effects of psychotropic drugs on the behavior and neurochemistry of olfactory bulbectomized rats. *Pharmacological Therapeutics* 47:21–34. [WJP]

Varela, F., Lachaux, J. P., Rodriguez, E. & Martinerie, J. (2001) The brainweb: Phase synchronization and large-scale integration. *Nature Reviews Neuroscience* 2(4):229–39. [SMD]

Vetter, P., Goodbody, S. J. & Wolpert, D. M. (1999) Evidence for an eye-centered spherical representation of the visuomotor map. *Journal of Neurophysiology* 81:935–39. [aBM]

von Holst, E. & Mittelstaedt, H. (1950) Das Reafferenzprinzip (Wechselwirkungen zwischen Zentralnervensystem und Peripherie). *Naturwissenschaften* 37: 464–76. [aBM]

Vroomen, J. & de Gelder, B. (2003) Visual motion influences the contingent auditory motion aftereffect. *Psychological Science* 14:357–61. [EM]

Waddi, P., Dickinson, A. & Schultz, W. (2001) Dopamine responses comply with basic assumptions of formal learning theory. *Nature* 412:43–48. [FA]

Waltzman, D. M., Silakov, V. L, DePalma-Bowles, S. & Ayers, A. S. (2006) Effects of reversible inactivation of the primate mesencephalic reticular formation. II. Hypometric vertical saccades. *Journal of Neurophysiology* 83:2265–99. [aBM]

Waleszczyk, W. J., Wang, C., Burke, W. & Dreher, B. (1999) Velocity response profiles of collicular neurons: Parallel and convergent visual information channels. *Neuroscience* 93:1063–76. [rBM]

Walker, M. P., Ayre, G. A., Cummings, J. L., Wesnes, K., McKeith, I. G., O'Brien, J. T. & Ballard, C. G. (2000) Quantifying fluctuation in dementia with Lewy bodies, Alzheimer's disease and Vascular dementia. *Neurology* 54:1616–25. [DC]

Wall, P. D. (1996) Response to editorial by Anand and Craig. *Pain* 66:209. [KJSA]

(1997) Do fetuses feel pain? Definition of pain needs clarification. *British Medical Journal* 314(7088):1201. [KJSA]

Wallace, S. F., Rosenquist, A. C. & Sprague, J. M. (1989) Recovery from cortical blindness mediated by destruction of nontectotectal fibers in the commissure of the superior colliculus in the cat. *Journal of Comparative Neurology* 284:429–50. [aBM]

(1990) Ibotenic acid lesions of the lateral substantia nigra restore visual orientation behavior in the hemianopic cat. *Journal of Comparative Neurology* 296:222–52. [aBM]

Wang, S. & Redgrave, P. (1997) Microinjections of muscimol into lateral superior colliculus disrupt orienting and oral movements in the formalin model of pain. *Neuroscience* 81:967–88. [aBM]

Wang, S., Wang, H., Niemi-Junkola, U., Westby, C. W. M., McHaffie, J. G., Stein, B. E. & Redgrave, P. (2000) Parallel analyses of nociceptive neurons in rat superior colliculus by using c-fos immunohistochemistry and electrophysiology under different conditions of anaesthesia. *Journal of Comparative Neurology* 425:599–615. [rBM]

Watkins, S., Shams, L., Tanaka, S., Haynes, J. D. & Rees, G. (2006) Sound alters activity in human V1 in association with illusory visual perception. *Neuroimage* 31(3):1247–56. [aBM, SW]

Watt, D. F. (1998) Affective neuroscience and extended reticular thalamic activating system theories of consciousness. Target Article, Association for the Scientific Study of Consciousness (ASSC) Electronic Seminar. http://server.phil.vt.edu/assc/esem4.html. [DFW]

(2000) The centrencephalon and thalamocortical integration: Neglected contributions of periaqueductal gray. *Consciousness and Emotion* 1:91–114. [aBM]

Watt, D. F. & Pinnus, D. L. (2004) Neural substrates of consciousness: Implications for clinical psychiatry. In: *Textbook of biological psychiatry*, ed. J. Panksepp, pp. 75–110. Wiley. [aBM, JP, DFW]

Webster, M. J., Bachevalier, J. & Ungerleider, L. G. (1993) Subcortical connections of inferior temporal areas TE and TEO in macaque monkeys. *Journal of Comparative Neurology* 335:73–91. [aBM]

Webster's Third New International Dictionary of the English language unabridged (1961) ed. P. Babcock Gove. 1993 reprint. Könemann. [aBM]

Weddell, R. A. (2004) Subcortical modulation of spatial attention including evidence that the Sprague effect extends to man. *Brain and Cognition* 55: 497–507. [arBM, SW]

Weiskrantz, L. (1996) Blindsight revisited. *Current Opinion in Neurobiology* 6:215–20. [R-PB]

(1997) *Consciousness lost and found: A neuropsychological exploration*. Oxford University Press. [SW]

(2004) Blindsight. In: *The Oxford companion to the mind*, ed. R. L. Gregory, pp. 108–110. Oxford University Press. [RAG]

Weiskrantz, L., Sanders, M. D. & Marshall, J. (1974) Visual capacity in the hemianopic field following a restricted cortical ablation. *Brain* 97:709–28. [JS]

Weller, R. E. (1988) Vision within extrageniculate striate systems. *Progress in Brain Research* 75:293–306. [rBM]

Wentzel, P. R., De Zeeuw, C. I., Holstege, J. C. & Gerrits, N. M. (1995) Inhibitory synaptic inputs to the oculomotor nucleus from vestibulo-ocular-reflex-related nuclei in the rabbit. *Neuroscience* 65:161–74. [aBM]

Werner, W., Dannenberg, S. & Hoffmann, P. (1997) Arm-movement-related neurons in the primate superior colliculus and underlying reticular formation: Comparison of neuronal activity with EMGs of muscles of the shoulder, arm and trunk during reaching. *Experimental Brain Research* 115:191–205. [aBM]

Whishaw, I. Q. (1990) The decorticate rat. In: *The cerebral cortex of the rat*, ed. B. Kolb & R. C. Tees, pp. 239–67. MIT Press. [aBM]

Whishaw, I. Q. & Kolb, B. (1983) Can male decorticate rats copulate? *Behavioral Neuroscience* 97:370–78. [rBM]

(1995) The mating movements of male decorticate rats: Evidence for subcortically generated movements by the male but regulation of approaches by the female. *Behavioural Brain Research* 17:171–91. [aBM]

(1988) Sparing of skilled forelimb reaching and corticospinal projections after neonatal motor cortex removal or hemidecortication in the rat: Support for the Kennard doctrine. *Brain Research* 451:97–114. [aBM]

Whishaw, I. Q., Schallert, T. & Kolb, B. (1981) An analysis of feeding and sensorimotor abilities of rats after decortication. *Journal of Comparative and Physiological Psychology* 95:85–103. [rBM]

Wiberg, M. (1992) Reciprocal connections between the periaqueductal gray matter and other somatosensory regions of the cat midbrain: A possible mechanism of pain inhibition. *Uppsala Journal of Medical Science* 97:37–47. [aBM]

Wicht, H. (1996) The brains of lampreys and hagfishes: Characteristics, characters, and comparisons. *Brain, Behavior and Evolution* 48:248–61. [aBM]

Wicht, H. & Northcutt, R. G. (1992) The forebrain of the pacific hagfish: A cladistic reconstruction of the ancestral craniate forebrain. *Brain, Behavior and Evolution* 40:25–64. [aBM]

Williams, C. (2005) Framing the fetus in medical work: Rituals and practices. *Social Science and Medicine* 60(9):2085–95. [KJSA]

Wilson, S. A. K. (1925) Disorders of motility and muscle tone with special reference to the corpus striatum (Croonian Lectures). *Lancet* 2:215–91. [TJP]

Windle, W. F. & Baxter, R. E. (1936) Development of reflex mechanisms in the spinal cord of albino rat embryos. Correlations between structure and function, and comparisons with the cat and the chick. *Journal of Comparative Neurology* 63:189–209. [aBM]

Winkielman, P. & Berridge, K. (2004) Unconscious emotion. *Current Directions in Psychological Science* 13:120–23. [CI]

Winn, P. (1998) Frontal syndrome as a consequence of lesions in the pedunculopontine tegmental nucleus: A short theoretical review. *Brain Research Bulletin* 47:551–63. [aBM]

Wintour, E. M., Lowitt, M., McFarlane, A., Moritz, K., Potocnik, S., Ross, S. & Tangalakis, K. (1996) Experimental hydranencephaly in the ovine fetus. *Acta Neuropathologica (Berlin)* 91:537–44. [aBM]

Woods, J. W. (1964) Behavior of chronic decerebrate rats. *Journal of Neurophysiology* 27:635–44. [aBM]

Wunderlich, K., Schneider, K. A. & Kastner, S. (2005) Neural correlates of binocular rivalry in the human lateral geniculate nucleus. *Nature Neuroscience* 8(11):1595–602. [SW]

Wurtz, R. H. & Albano, J. E. (1980) Visual-motor function of the primate superior colliculus. *Annual Review of Neuroscience* 3:189–226. [FA]

Wurtz, R. H. & Mohler, C. W. (1974) Selection of visual targets for the initiation of saccadic eye movements. *Brain Research* 71:209–14. [aBM]

Yamashita, M., Mori, T., Nagata, K., Yeh, J. Z. & Narahashi, T. (2005) Isoflurane modulation of neuronal nicotinic acetylcholine receptors expressed in human embryonic kidney cells. *Anesthesiology* 103:76–84. [DC]

Yarrow, K., Johnson, H., Haggard, P. & Rothwell, J. C. (2004) Consistent chronostasis effects across saccade categories imply a subcortical efferent trigger. *Journal of Cognitive Neuroscience* 16:839–47. [aBM]

Yuge, T. & Kaga, K. (1998) Brain functions of an infant with hydranencephaly revealed by auditory evoked potentials. *International Journal of Pediatric Otorhinolaryngology* 45:91–95. [aBM]

Zadeh, L. A. & Kacprzyk, J., eds. (1992) *Fuzzy logic for the management of uncertainty*. Wiley. [RAG]

Zahavi, D. (2005) *Subjectivity and selfhood*. MIT Press. [GN]

Zahm, D. S. (2006) The evolving theory of basal forebrain functional-anatomical "macrosystems." *Neuroscience and Biobehavioral Reviews* 30:148–72. [aBM]

Zajonc, R. B. (1980) Feeling and thinking: Preferences need no inferences. *American Psychologist* 35:151–75. [CI]

Zelazo, P. D. (1999) Language, levels of consciousness, and the development of intentional action. In: *Developing theories of intention: Social understanding and self-control*, ed. P. D. Zelazo, J. W. Astington & D. R. Olson, pp. 95–117. Erlbaum. [AM]

(2004) The development of conscious control in childhood. *Trends in Cognitive Sciences* 8(1):12–17. [KJSA]

Zella, J. C., Brugge, J. F. & Schnupp, J. W. H. (2001) Passive eye displacement alters auditory spatial receptive fields of cat superior colliculus neurons. *Nature Neuroscience* 4:1167–69. [aBM]

Zeman, A. (2001) Consciousness. *Brain* 124:1263–89. [AMLC]

Zhang, K., Crady, C. J., Tsapakis, E. M., Andersen, S. L., Tarazi, F. I. & Baldessarini, R. J. (2004) Regulation of working memory by dopamine D4 receptor in rats. *Neuropsychopharmacology* 29:1645–55. [FA]

Zhu, J. J. & Lo, F. S. (2000) Recurrent inhibitory circuitry in the deep layers of the rabbit superior colliculus. *Journal of Physiology* 523:731–49. [aBM]

Zlatkova, I., Ballard, C. G., Aarsland, D., Larsen, J. P., McKeith, I. G., Perry, R. H. & Perry, E. K. (2006) Lewy body disease: Thalamic cholinergic activity related to dementia and parkinsonism. *Neurobiology of Aging* 27:433–38. [DC]

Zompa, I. C. & Dubuc, R. (1996) A mesencephalic relay for visual inputs to reticulospinal neurones in lampreys. *Brain Research* 718:221–27. [aBM]

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RCOG ARTICLE ON FETAL PAIN IS A "STUNNING LACK OF SCHOLARSHIP"
Attempt by abortion advocates to mislead the public

PITTSBURGH, PA – As the 40th Annual National Right to Life Convention continued with a special general session focusing on the pain of the unborn child, a Working Party of the Royal College of Obstetricians and Gynaecologists (RCOG) released an article disputing an overwhelming body of evidence that unborn children can feel pain *in utero*.

The following statement may be attributed to National Right to Life Director of State Legislation Mary Spaulding Balch, J.D.

> **An objective expert in neurobiology would be appalled by the stunning lack of scholarship in the RCOG article. Its authors (predominantly abortion advocates and at least one abortionist) based their claim that unborn children do not experience pain before 24 weeks on the absence of complete nerve connection to the cortex before then.**
>
> **They ignore the seminal 2007 publication of "Consciousness without a cerebral cortex," in the medical journal *Behavioral and Brain Sciences* and dismiss its evidence that children born missing virtually all of the cerebral cortex nonetheless experience pain.**
>
> **Ironically, the article concedes the evidence that by 20 weeks pain receptors are present throughout the unborn child's skin, that these are linked by nerves to the thalamus and the subcortal plate, and that these children have coordinated aversive reactions to painful stimuli, and experience increased stress hormones from it.**
>
> **This article is an effort by acknowledged abortion promoters to mislead the public at-large – and most tragically women considering abortion – about the increasing evidence demonstrating the unborn child's sensitivity to pain.**

The issue of fetal pain has captured headlines thanks to a landmark law enacted by the Nebraska legislature in April which restricts abortion after twenty weeks declaring that the state has a compelling interest in the life of a pain-capable unborn child at and after twenty weeks.

The 2007 article from* Behavioral and Brain Sciences *is available from the NRLC Communications Department. NRLC's Balch is available to discuss the issue of fetal pain, the RCOG article, and the body of research demonstrating that unborn children are capable of feeling pain. To arrange an interview contact the NRLC Communications Department on-site at the 40th Annual National Right to Life Convention at (724) 899-6245.

The National Right to Life Committee, the nation's largest pro-life group is a federation of 50 state right-to-life affiliates and more than 3,000 local chapters.

http://www.nrlc.org/abortion/fetal_pain/nrlcrebuttaljama.html

NATIONAL RIGHT TO LIFE: GULLIBLE TREATMENT OF TRUMPED UP "STUDY" ON FETAL PAIN ISSUE SHOULD EMBARRASS J.A.M.A. AND SOME JOURNALISTS

This is an update from the National Right to Life Committee, 202-626-8825, issued Thursday, August 25, 2005, at 4 PM EDT. For further updates on this subject, watch http://www.nrlc.org/abortion/fetal_pain/index.html

This memo offers a number of points of information regarding the article "Fetal Pain: A Systematic Multidisciplinary Review of the Evidence," published in the August 24 edition of the Journal of the American Medical Association (JAMA). Any of the material below, if not otherwise attributed, can be attributed to NRLC Legislative Director Douglas Johnson (Legfederal@aol.com), who prepared this memorandum.

BASIC OBJECTIONS

1. The JAMA article was produced by pro-abortion activists. There is no new laboratory research reported in the article -- it is merely a commentary on a selection of existing medical literature. The authors purport to show that there is no good evidence that human fetuses feel pain before 29 weeks (during the seventh month). The authors' conclusion (which was predetermined by their political agenda -- see below) is disputed by experts with far more extensive credentials in pain research than any of the authors. These independent authorities say that there is substantial evidence from multiple lines of research that unborn humans can perceive pain during the fifth and sixth months (i.e., by 20 weeks gestational age), and perhaps somewhat earlier.

2. For example, Dr. Kanwaljeet S. Anand, a pain researcher who holds tenured chairs in pediatrics, anesthesiology, pharmacology, and neurobiology at the University of Arkansas, said in a document accepted as expert by a federal court, "It is my opinion that the human fetus possesses the ability to experience pain from 20 weeks of gestation, if not earlier, and that pain perceived by a fetus is possibly more intense than that perceived by newborns or older children." Read Dr. Anand's complete statement entered in federal court, summarizing the scientific evidence, here. In a USA Today article (August 25), Dr. Anand predicted that JAMA's publication of the article would "inflame a lot of scientists who are . . . far more knowledgeable in this area than the authors appear to be."

3. A similar review published in September 1999 in the British Journal of Obstetrics and Gynaecology (the leading ob-gyn journal in the UK) concluded: "Given the anatomical evidence, it is possible that the fetus can feel pain from 20 weeks and is caused distress by interventions from as early as 15 or 16 weeks." (Article available in PDF format here.)

4. The JAMA authors arrive at their "conclusion" through a highly tendentious methodology that could, for the most part, also be used to argue that there is no proof that animals really feel pain and no proof that premature newborn humans really feel pain

(although the authors do not address those subjects). There are innumerable state and federal laws intended to reduce the suffering of animals, even though it is impossible to "prove" that their "experience" of pain is subjectively the same as that of the lawmakers who have enacted these regulations.

THE EVIDENCE FROM PREMATURELY BORN INFANTS

5. Infants born as early as 23 or 24 weeks now commonly survive long term in neonatal intensive care units. Neonatologists confirm that they react negatively to painful stimuli -- for example, by grimacing, withdrawing, and whimpering. When they must receive surgical procedures, they are given drugs to prevent pain. Yet, the JAMA authors assert that there is no credible evidence of fetal pain until 29 weeks -- which is five or six weeks later. If these babies feel pain in the incubator, then they also feel pain in the womb. If the newborn at 23 weeks demonstrates aversion to pain and needs protection from pain, the same is true of the 24-week (or 25-week, 26-week, 27-week, or 28-week) unborn child.

6. As Dr. Paul Ranalli, a neurologist at the University of Toronto, commented on the paper: "Across the nation, Neonatal Intensive Care Units (NICUs) are full of bravely struggling preemies . . . The only difference between a child in the womb at this stage, or one born and cared for in an incubator, is how they receive oxygen -- either through the umbilical cord or through the lungs. There is no difference in their nervous systems. Their article sets back humane pediatric medicine 20 years, back to a time when doctors still believed babies could not feel pain." In testimony before a congressional committee in 1996, Dr. Jean A. Wright, then a pediatric pain specialist at Emory University, said: "Preterm infants who are born and delivered at 23 weeks of gestation show very highly specific and well-coordinated physiologic and behavioral responses to pain which is just like older infants." (Even the paper notes in passing, "Normal EEG patterns have been characterized for neonates as young as 24 weeks' postconceptual age.")

THE VIOLENCE OF ABORTION METHODS USED

7. The gross trauma inflicted on the unborn human by abortion methods used in the fifth and sixth months far exceed anything that would be done to a premature newborn at the same stage of development. The most common abortion method, the so-called "D&E," involves tearing arms and legs off of the unanesthetized unborn child, then crushing the skull. (Click here to see a series of professional medical school illustrations of this method.) Thousands of times annually, the partial-birth abortion method is used, which involves mostly delivering the living premature infant, feet first, and then puncturing the skull with scissors or a pointed metal tube (to see medically accurate illustrations of this method, click here). To review material presented to Congress by leading anesthesiologists and other medical experts with varying positions on legal abortion, click here.

THE ORIGINS OF THE PAPER

8. The so-called "study" was produced by pro-abortion activists and a well-known practitioner of late abortions -- but, with a few notable exceptions, that readily available information was omitted or greatly minimized by mainstream media outlets that initially covered story on August 23 and 24, including ABC World News Tonight, the Associated Press, and the New York Times.

9. The lead author of the article, Susan J. Lee, who is now a medical student, was previously employed as a lawyer by NARAL, the pro-abortion political advocacy organization (Knight Ridder, August 24).

10. One of Lee's four co-authors, Dr. Eleanor A. Drey, is the director of the largest abortion clinic in San Francisco (San Francisco Chronicle, March 31, 2004, and Knight Ridder, August 24, 2005). According to Dr. Drey, the abortion facility that she runs performs about 600 abortions a year between the 20th and 23rd weeks of pregnancy (i.e., in the fifth and sixth months). (San Francisco Chronicle, March 31, 2004) Drey is a prominent critic of the Partial-Birth Abortion Ban Act, and a self-described activist. (In a laudatory profile in the newsletter of Physicians for Reproductive Choice and Health, September 2004, it was noted that "much of Dr. Drey's research centers on repeat and second-trimester procedures . . .," and quotes Drey as saying, "I am very lucky because I get to train residents and medical students, and I really do feel that it's a type of activism.") Drey is also on the staff of the Center for Reproductive Health Research and Policy (CRHRP) at the University of California, San Francisco -- a pro-abortion propaganda and training center. Much of this information was available through even a very cursory Google search, and some of it was provided to journalists who contacted NRLC about the embargoed JAMA paper on August 22-23, but few saw fit to mention these connections in their initial reports.

11. However, one reporter (Knight Ridder's Marie McCullough) did contact JAMA editor-in-chief Catherine D. DeAngelis regarding the ties of Lee and Drey. McCullough reported that DeAngelis "said she was unaware of this, and acknowledged it might create an appearance of bias that could hurt the journal's credibility. 'This is the first I've heard about it,' she said. 'We ask them to reveal any conflict of interest. I would have published' the disclosure if it had been made." (Knight Ridder, August 24, 2005) A day later, DeAngelis told USA Today that the affiliations of Drey and Lee "aren't relevant," but again said that the ties should have been disclosed. If she really thought the affiliations were not relevant, why would she say that they should have been disclosed? If a review of the same issue by doctors employed by pro-life advocacy groups had been submitted or published, would those affiliations have been ignored by journalists?

12. Dr. David Grimes, a vice-president of Family Health International, has been relied on by CNN, the New York Times, and some other media as a purported expert to defend the paper. Dr. Grimes has made pro-abortion advocacy a central element of his career for decades. (During the time he worked for the CDC in the 1980s, his off-hours work at a local late-abortion facility sparked protests from some pro-life activists. In 1987, a year

after he left the CDC, Grimes testified that he had already performed more than 10,000 abortions, 10 to 20 percent of those after the first trimester.) In addition, Grimes was previously the chief of the Department of Obstetrics, Gynecology and Reproductive Sciences at the San Francisco General Hospital -- the very same institution where author Drey directs the abortion clinic.

THE FINDINGS OF A FEDERAL COURT

13. In 2004, the U.S. District Court for the Southern District of New York received extensive testimony regarding fetal pain from experts on both sides, including doctors who perform many late abortions, as part of a legal challenge to the Partial-Birth Abortion Ban Act. Although the subsequent opinion struck down the ban as inconsistent with a 2000 U.S. Supreme Court ruling (this is being appealed), the court made certain formal "findings of fact," among these: "The Court finds that the testimony at trial and before Congress establishes that D&X [partial-birth abortion] is a gruesome, brutal, barbaric, and uncivilized medical procedure. Dr. Anand's testimony, which went unrebutted by Plaintiffs, is credible evidence that D&X abortions subject fetuses to severe pain. Notwithstanding this evidence, some of Plaintiffs' experts testified that fetal pain does not concern them, and that some do not convey to their patients that their fetuses may undergo severe pain during a D&X." (This illustrates that abortionists will not raise the question of pain, at any stage of pregnancy, unless they are required to do so.)

UNBORN CHILD PAIN AWARENESS ACT (S. 51, H.R. 356)

14. The obvious purpose of the authors of the JAMA paper was to damage the prospects for the Unborn Child Pain Awareness Act (S. 51, H.R. 356). This bill would require that abortion providers give women seeking abortions after 20 weeks after fertilization (22 weeks gestation) certain basic information on the substantial evidence that their unborn children may experience pain while being aborted, and advise them regarding any available methods to reduce or eliminate such pain. The bill explicitly states that the abortion provider may offer his or her own opinions and advice regarding the question, including discussion of any risks to the mother of methods of reducing the pain of the unborn child. The authors, in their final paragraph, explicitly oppose any requirement that abortionists raise the pain issue in any fashion, at least during the fifth and sixth months.

15. It is noteworthy, however, that in January, 2005, NARAL President Nancy Keenan issued a statement that NARAL "does not intend to oppose" the bill, because "pro-choice Americans have always believed that women deserve access to all the information relevant to their reproductive health decisions." (A complete reproduction of the NARAL statement is available here.)

16. Spokepersons for some groups of abortion providers say that they object to the Unborn Child Pain Awareness Act because it would require that abortionists recite a "script" advising women who are seeking abortions after 22 weeks gestational age (20

weeks from fertilization) that there is "substantial evidence" that abortion will inflict pain (the bill also explicitly says that the abortionist may also offer whatever opinions he or she wishes regarding the issue and the risks of any optional pain relieving methods). But in truth, abortion providers, like the authors of the paper, object not just to a "script" but to any requirement whatever that women be provided with any information on the subject. They have also objected to laws enacted in Arkansas and Georgia that require only the provision of printed information prepared by the state health agencies, and to a Minnesota law that merely requires that the abortionist tell the woman "whether or not an anesthetic or analgesic would eliminate or alleviate organic pain to the unborn child caused by the particular method of abortion to be employed and the particular medical benefits and risks associated with the particular anesthetic or analgesic." Apparently, the abortionists are taking the paternalistic stance that women are incapable of evaluating such information and giving it whatever weight they think it deserves.

ADMINISTERING ANESTHESIA OR ANALGESICS

17. The authors of the JAMA paper say that "no established protocols exist for administering anesthesia or analgesia directly to the fetus for minimally invasive fetal procedures or abortions." (p. 952) Yet, some abortions are performed by administering toxins into the amniotic sac (or even directly into the fetal heart) with a needle, precisely guided by ultrasound. Moreover, in cases of women carrying multiple unborn humans, abortionists sometimes engage in "selective reduction," in which some of the fetuses are killed by stabbing them directly in their hearts with a needle guided by ultrasound. One suspects, therefore, that any current lack of methods of safely administering pain-reducing drugs to a fetus in utero relate more the fact that abortionists just don't care about fetal pain and have not developed such methods, rather than to any insurmountable technical obstacles. In any case, under the Unborn Child Pain Awareness Act, a woman considering an abortion after 20 weeks gestational age would be given information on the current state of the art, including the abortionist's own assessment of any risks, to evaluate as she sees fit.

18. Paul Ranalli, a neurologist at the University of Toronto, reports, "Experts from Britain and France have proposed safe and effective fetal anesthesia protocols. (Ranalli cites the 1997 Working Party Report on Fetal Pain by the UK's Royal College of Obstetrics and Gynecology and "La douleur du foetus," Mahieu-Caputo D, Dommergues M et al, Presse Med 2000; 29:663-9, recommending Sulfentanyl 1 ug/kg and Pentothal 10 ug/kg.) Ranalli also writes that the JAMA paper itself "includes experimental animal evidence that suggests an effective intra-amniotic needle injection could spare the fetus pain, without the need to give the mother any additional anesthetic" (citing material on JAMA p. 952, column 1).

NUMBERS OF ABORTION AT ISSUE

19. According to the JAMA paper, relying on a CDC report, about 1.4 percent of the abortions performed in the U.S. are performed at or after 21 weeks gestational age. If so, that would be over 18,000 abortions annually nationwide -- hardly inconsequential to

anyone concerned with inflicting pain on a sentient young human. (Note: That figure omits abortions performed at 20 weeks gestational age.) It is worth noting that the CDC reports are very incomplete. Indeed, the report itself makes it clear that the CDC received no abortion reports from California -- so none of the 600 abortions performed annually at 20-23 weeks in Dr. Drey's abortion clinic are reflected in the CDC figures.

February 10, 2008

The First Ache

By ANNIE MURPHY PAUL

Twenty-five years ago, when Kanwaljeet Anand was a medical resident in a neonatal intensive care unit, his tiny patients, many of them preterm infants, were often wheeled out of the ward and into an operating room. He soon learned what to expect on their return. The babies came back in terrible shape: their skin was gray, their breathing shallow, their pulses weak. Anand spent hours stabilizing their vital signs, increasing their oxygen supply and administering insulin to balance their blood sugar.

"What's going on in there to make these babies so stressed?" Anand wondered. Breaking with hospital practice, he wrangled permission to follow his patients into the O.R. "That's when I discovered that the babies were not getting anesthesia," he recalled recently. Infants undergoing major surgery were receiving only a paralytic to keep them still. Anand's encounter with this practice occurred at John Radcliffe Hospital in Oxford, England, but it was common almost everywhere. Doctors were convinced that newborns' nervous systems were too immature to sense pain, and that the dangers of anesthesia exceeded any potential benefits.

Anand resolved to find out if this was true. In a series of clinical trials, he demonstrated that operations performed under minimal or no anesthesia produced a "massive stress response" in newborn babies, releasing a flood of fight-or-flight hormones like adrenaline and cortisol. Potent anesthesia, he found, could significantly reduce this reaction. Babies who were put under during an operation had lower stress-hormone levels, more stable breathing and blood-sugar readings and fewer postoperative complications. Anesthesia even made them more likely to survive. Anand showed that when pain relief was provided during and after heart operations on newborns, the mortality rate dropped from around 25 percent to less than 10 percent. These were extraordinary results, and they helped change the way medicine is practiced. Today, adequate pain relief for even the youngest infants is the standard of care, and the treatment that so concerned Anand two decades ago would now be considered a violation of medical ethics.

But Anand was not through with making observations. As NICU technology improved, the preterm infants he cared for grew younger and younger — with gestational ages of 24 weeks, 23, 22 — and he noticed that even the most premature babies grimaced when pricked by a needle. "So I said to myself, Could it be that this pain system is developed and functional before the baby is born?" he told me in the fall. It was not an abstract question: fetuses as well as newborns may now go under the knife. Once highly experimental, fetal surgery — to remove lung tumors, clear blocked urinary tracts, repair malformed diaphragms — is a frequent occurrence at a half-dozen fetal treatment centers around the country, and could soon become standard care for some conditions diagnosed prenatally like spina bifida. Whether the fetus feels pain is a question that matters to the doctor wielding the scalpel.

And it matters, of course, for the practice of abortion. Over the past four years, anti-abortion groups have turned fetal pain into a new front in their battle to restrict or ban abortion. Anti-abortion politicians have drafted laws requiring doctors to tell patients seeking abortions that a fetus can feel pain and to offer the fetus anesthesia; such legislation has already passed in five states. Anand says he does not oppose abortion

in all circumstances but says decisions should be made on a case-by-case basis. Nonetheless, much of the activists' and lawmakers' most powerful rhetoric on fetal pain is borrowed from Anand himself.

Known to all as Sunny, Anand is a soft-spoken man who wears the turban and beard of his Sikh faith. Now a professor at the University of Arkansas for Medical Sciences and a pediatrician at the Arkansas Children's Hospital in Little Rock, he emphasizes that he approaches the question of fetal pain as a scientist: "I eat my best hypotheses for breakfast," he says, referring to the promising leads he has discarded when research failed to bear them out. New evidence, however, has persuaded him that fetuses can feel pain by 20 weeks gestation (that is, halfway through a full-term pregnancy) and possibly earlier. As Anand raised awareness about pain in infants, he is now bringing attention to what he calls "signals from the beginnings of pain."

But these signals are more ambiguous than those he spotted in newborn babies and far more controversial in their implications. Even as some research suggests that fetuses can feel pain as preterm babies do, other evidence indicates that they are anatomically, biochemically and psychologically distinct from babies in ways that make the experience of pain unlikely. The truth about fetal pain can seem as murky as an image on an ultrasound screen, a glimpse of a creature at once recognizably human and uncomfortably strange.

IF THE NOTION that newborns are incapable of feeling pain was once widespread among doctors, a comparable assumption about fetuses was even more entrenched. Nicholas Fisk is a fetal-medicine specialist and director of the University of Queensland Center for Clinical Research in Australia. For years, he says, "I would be doing a procedure to a fetus, and the mother would ask me, 'Does my baby feel pain?' The traditional, knee-jerk reaction was, 'No, of course not.' " But research in Fisk's laboratory (then at Imperial College in London) was making him uneasy about that answer. It showed that fetuses as young as 18 weeks react to an invasive procedure with a spike in stress hormones and a shunting of blood flow toward the brain — a strategy, also seen in infants and adults, to protect a vital organ from threat. Then Fisk carried out a study that closely resembled Anand's pioneering research, using fetuses rather than newborns as his subjects. He selected 45 fetuses that required a potentially painful blood transfusion, giving one-third of them an injection of the potent painkiller fentanyl. As with Anand's experiments, the results were striking: in fetuses that received the analgesic, the production of stress hormones was halved, and the pattern of blood flow remained normal.

Fisk says he believes that his findings provide suggestive evidence of fetal pain — perhaps the best evidence we'll get. Pain, he notes, is a subjective phenomenon; in adults and older children, doctors measure it by asking patients to describe what they feel. ("On a scale of 0 to 10, how would you rate your current level of pain?") To be certain that his fetal patients feel pain, Fisk says, "I would need one of them to come up to me at the age of 6 or 7 and say, 'Excuse me, Doctor, that bloody hurt, what you did to me!' " In the absence of such first-person testimony, he concludes, it's "better to err on the safe side" and assume that the fetus can feel pain starting around 20 to 24 weeks.

Blood transfusions are actually among the least invasive medical procedures performed on fetuses. More intrusive is endoscopic fetal surgery, in which surgeons manipulate a joystick-like instrument while watching the fetus on an ultrasound screen. Most invasive of all is open fetal surgery, in which a pregnant woman's uterus is cut open and the fetus exposed. Ray Paschall, an anesthesiologist at Vanderbilt Medical Center in Nashville, remembers one of the first times he provided anesthesia to the mother and minimally to the fetus in an open fetal operation, more than 10 years ago. When the surgeon lowered his scalpel to the 25-week-old fetus, Paschall saw the tiny figure recoil in what looked to him like pain. A few months later, he watched another fetus, this one 23 weeks old, flinch at the touch of the instrument. That was enough for

Paschall. In consultation with the hospital's pediatric pain specialist, "I tremendously upped the dose of anesthetic to make sure that wouldn't happen again," he says. In the more than 200 operations he has assisted in since then, not a single fetus has drawn back from the knife. "I don't care how primitive the reaction is, it's still a human reaction," Paschall says. "And I don't believe it's right. I don't want them to feel pain."

But whether pain is being felt is open to question. Mark Rosen was the anesthesiologist at the very first open fetal operation, performed in 1981 at the University of California, San Francisco, Medical Center, and the fetal anesthesia protocols he pioneered are now followed by his peers all over the world. Indeed, Rosen may have done more to prevent fetal pain than anyone else alive — except that he doesn't believe that fetal pain exists. Research has persuaded him that before a point relatively late in pregnancy, the fetus is unable to perceive pain.

Rosen provides anesthesia for a number of other important reasons, he explains, including rendering the pregnant woman unconscious and preventing her uterus from contracting and setting off dangerous bleeding or early labor. Another purpose of anesthesia is to immobilize the fetus during surgery, and indeed, the drugs Rosen supplies to the pregnant woman do cross the placenta to reach the fetus. Relief of fetal pain, however, is not among his objectives. "I have every reason to want to believe that the fetus feels pain, that I've been treating pain all these years," says Rosen, who is intense and a bit prickly. "But if you look at the evidence, it's hard to conclude that that's true."

Rosen's own hard look at the evidence came a few years ago, when he and a handful of other doctors at U.C.S.F. pulled together more than 2,000 articles from medical journals, weighing the accumulated evidence for and against fetal pain. They published the results in The Journal of the American Medical Association in 2005. "Pain perception probably does not function before the third trimester," concluded Rosen, the review's senior author. The capacity to feel pain, he proposed, emerges around 29 to 30 weeks gestational age, or about two and a half months before a full-term baby is born. Before that time, he asserted, the fetus's higher pain pathways are not yet fully developed and functional.

What about a fetus that draws back at the touch of a scalpel? Rosen says that, at least early on, this movement is a reflex, like a leg that jerks when tapped by a doctor's rubber mallet. Likewise, the release of stress hormones doesn't necessarily indicate the experience of pain; stress hormones are also elevated, for example, in the bodies of brain-dead patients during organ harvesting. In order for pain to be felt, he maintains, the pain signal must be able to travel from receptors located all over the body, to the spinal cord, up through the brain's thalamus and finally into the cerebral cortex. The last leap to the cortex is crucial, because this wrinkly top layer of the brain is believed to be the organ of consciousness, the generator of awareness of ourselves and things not ourselves (like a surgeon's knife). Before nerve fibers extending from the thalamus have penetrated the cortex — connections that are not made until the beginning of the third trimester — there can be no consciousness and therefore no experience of pain.

Sunny Anand reacted strongly, even angrily, to the article's conclusions. Rosen and his colleagues have "stuck their hands into a hornet's nest," Anand said at the time. "This is going to inflame a lot of scientists who are very, very concerned and are far more knowledgeable in this area than the authors appear to be. This is not the last word — definitely not." Anand acknowledges that the cerebral cortex is not fully developed in the fetus until late in gestation. What is up and running, he points out, is a structure called the subplate zone, which some scientists believe may be capable of processing pain signals. A kind of holding station for developing nerve cells, which eventually melds into the mature brain, the subplate zone becomes

operational at about 17 weeks. The fetus's undeveloped state, in other words, may not preclude it from feeling pain. In fact, its immature physiology may well make it more sensitive to pain, not less: the body's mechanisms for inhibiting pain and making it more bearable do not become active until after birth.

The fetus is not a "little adult," Anand says, and we shouldn't expect it to look or act like one. Rather, it's a singular being with a life of the senses that is different, but no less real, than our own.

THE SAME MIGHT be said of the five children who were captured on video by a Swedish neuroscientist named Bjorn Merker on a trip to Disney World a few years ago. The youngsters, ages 1 to 5, are shown smiling, laughing, fussing, crying; they appear alert and aware of what is going on around them. Yet each of these children was born essentially without a cerebral cortex. The condition is called hydranencephaly, in which the brain stem is preserved but the upper hemispheres are largely missing and replaced by fluid.

Merker (who has held positions at universities in Sweden and the United States but is currently unaffiliated) became interested in these children as the living embodiment of a scientific puzzle: where consciousness originates. He joined an online self-help group for the parents of children with hydranencephaly and read through thousands of e-mail messages, saving many that described incidents in which the children seemed to demonstrate awareness. In October 2004, he accompanied the five on the trip to Disney World, part of an annual get-together for families affected by the condition. Merker included his observations of these children in an article, published last year in the journal Behavioral and Brain Sciences, proposing that the brain stem is capable of supporting a preliminary kind of awareness on its own. "The tacit consensus concerning the cerebral cortex as the 'organ of consciousness,' " Merker wrote, may "have been reached prematurely, and may in fact be seriously in error."

Merker's much-discussed article was accompanied by more than two dozen commentaries by prominent researchers. Many noted that if Merker is correct, it could alter our understanding of how normal brains work and could change our treatment of those who are now believed to be insensible to pain because of an absent or damaged cortex. For example, the decision to end the life of a patient in a persistent vegetative state might be carried out with a fast-acting drug, suggested Marshall Devor, a biologist at the Center for Research on Pain at Hebrew University in Jerusalem. Devor wrote that such a course would be more humane than the weeks of potentially painful starvation that follows the disconnection of a feeding tube (though as a form of active euthanasia it would be illegal in the United States and most other countries). The possibility of consciousness without a cortex may also influence our opinion of what a fetus can feel. Like the subplate zone, the brain stem is active in the fetus far earlier than the cerebral cortex is, and if it can support consciousness, it can support the experience of pain. While Mark Rosen is skeptical, Anand praises Merker's work as a "missing link" that could complete the case for fetal pain.

But anatomy is not the whole story. In the fetus, especially, we can't deduce the presence or absence of consciousness from its anatomical development alone; we must also consider the peculiar environment in which fetuses live. David Mellor, the founding director of the Animal Welfare Science and Bioethics Center at Massey University in New Zealand, says he was prompted to consider the role of fetal surroundings in graduate school. "Have you ever wondered," one visiting professor asked, "why a colt doesn't get up and gallop around inside the mare?" After all, a horse only minutes old is already able to hobble around the barnyard. The answer, as Mellor reported in an influential review published in 2005, is that biochemicals produced by the placenta and fetus have a sedating and even an anesthetizing effect on the fetus (both equine and human). This fetal cocktail includes adenosine, which suppresses brain activity; pregnanolone, which relieves pain; and prostaglandin D2, which induces sleep — "pretty potent stuff," he says.

Combined with the warmth and buoyancy of the womb, this brew lulls the fetus into a near-continuous slumber, rendering it effectively unconscious no matter what the state of its anatomy. Even the starts and kicks felt by a pregnant woman, he says, are reflex movements that go on in a fetus's sleep. While we don't know if the intense stimulation of surgery would wake it up, Mellor notes that when faced with other potential threats, like an acute shortage of oxygen, the fetus does not rouse itself but rather shuts down more completely in an attempt to conserve energy and promote survival. This is markedly different from the reaction of an infant, who will thrash about in an effort to dislodge whatever is blocking its airway. "A fetus," Mellor says, "is not a baby who just hasn't been born yet."

Even birth may not inaugurate the ability to feel pain, according to Stuart Derbyshire, a psychologist at the University of Birmingham in Britain. Derbyshire is a prolific commentator on the subject and an energetic provocateur. In milder moods, he has described the notion of fetal pain as a "fallacy"; when goaded by his critics' "lazy" thinking, he has pronounced it a "moral blunder" and "a shoddy, sentimental argument."

For all his vehemence in print, Derbyshire is affable in conversation, explaining that his laboratory research on the neurological basis of pain in adults led him to the matter of what fetuses feel: "For me, it's an interesting test case of what we know about pain. It's a great application of theory, basically." The theory, in this case, is that the experience of pain has to be learned — and the fetus, lacking language or interactions with caregivers, has no chance of learning it. In place of distinct emotions, it experiences a blur of sensations, a condition Derbyshire has likened to looking at "a vast TV screen with all of the world's information upon it from a distance of one inch; a great buzzing mass of meaningless information," he writes. "Before a symbolic system such as language, an individual will not know that something in front of them is large or small, hot or cold, red or green" — or, Derbyshire argues, painful or pleasant.

He finds "outrageous" the suggestion that the fetus feels anything like the pain that an older child or an adult experiences. "A fetus is biologically human, of course," he says. "It isn't a cow. But it's not yet psychologically human." That is a status not bestowed at conception but earned with each connection made and word spoken. Following this logic to its conclusion, Derbyshire has declared that babies cannot feel pain until they are 1 year old. His claim has become notorious in pain-research circles, and even Derbyshire says he thinks he may have overstepped. "I sometimes regret that I pushed it out quite that far," he concedes. "But really, who knows when the light finally switches on?"

IN FACT, "THERE may not be a single moment when consciousness, or the potential to experience pain, is turned on," Nicholas Fisk wrote with Vivette Glover, a colleague at Imperial College, in a volume on early pain edited by Anand. "It may come on gradually, like a dimmer switch." It appears that this slow dawning begins in the womb and continues even after birth. So where do we draw the line? When does a release of stress hormones turn into a grimace of genuine pain?

Recent research provides a potentially urgent reason to ask this question. It shows that pain may leave a lasting, even lifelong, imprint on the developing nervous system. For adults, pain is usually a passing sensation, to be waited out or medicated away. Infants, and perhaps fetuses, may do something different with pain: some research suggests they take it into their bodies, making it part of their fast-branching neural networks, part of their flesh and blood.

Anna Taddio, a pain specialist at the Hospital for Sick Children in Toronto, noticed more than a decade ago that the male infants she treated seemed more sensitive to pain than their female counterparts. This discrepancy, she reasoned, could be due to sex hormones, to anatomical differences — or to a painful event experienced by many boys: circumcision. In a study of 87 baby boys, Taddio found that those who had been

circumcised soon after birth reacted more strongly and cried for longer than uncircumcised boys when they received a vaccination shot four to six months later. Among the circumcised boys, those who had received an analgesic cream at the time of the surgery cried less while getting the immunization than those circumcised without pain relief.

Taddio concluded that a single painful event could produce effects lasting for months, and perhaps much longer. "When we do something to a baby that is not an expected part of its normal development, especially at a very early stage, we may actually change the way the nervous system is wired," she says. Early encounters with pain may alter the threshold at which pain is felt later on, making a child hypersensitive to pain — or, alternatively, dangerously indifferent to it. Lasting effects might also include emotional and behavioral problems like anxiety and depression, even learning disabilities (though these findings are far more tentative).

Do such long-term effects apply to fetuses? They may well, especially since pain experienced in the womb would be even more anomalous than pain encountered soon after birth. Moreover, the ability to feel pain may not need to be present in order for "noxious stimulation" — like a surgeon's incision — to do harm to the fetal nervous system. This possibility has led some to venture an early end to the debate over fetal pain. Marc Van de Velde, an anesthesiologist and pain expert at University Hospitals Gasthuisberg in Leuven, Belgium, says: "We know that the fetus experiences a stress reaction, and we know that this stress reaction may have long-term consequences — so we need to treat the reaction as well as we can. Whether or not we call it pain is, to me, irrelevant."

BUT THE QUESTION of fetal pain is not irrelevant when applied to abortion. On April 4, 2004, Sunny Anand took the stand in a courtroom in Lincoln, Neb., to testify as an expert witness in the case of Carhart v. Ashcroft. This was one of three federal trials held to determine the constitutionality of the ban on a procedure called intact dilation and extraction by doctors and partial-birth abortion by anti-abortion groups. Anand was asked whether a fetus would feel pain during such a procedure. "If the fetus is beyond 20 weeks of gestation, I would assume that there will be pain caused to the fetus," he said. "And I believe it will be severe and excruciating pain."

After listening to Anand's testimony and that of doctors opposing the law, Judge Richard G. Kopf declared in his opinion that it was impossible for him to decide whether a "fetus suffers pain as humans suffer pain." He ruled the law unconstitutional on other grounds. But the ban was ultimately upheld by the U.S. Supreme Court, and Anand's statements, which he repeated at the two other trials, helped clear the way for legislation aimed specifically at fetal pain. The following month, Sam Brownback, Republican of Kansas, presented to the Senate the Unborn Child Pain Awareness Act, requiring doctors to tell women seeking abortions at 20 weeks or later that their fetuses can feel pain and to offer anesthesia "administered directly to the pain-capable unborn child." The bill did not pass, but Brownback continues to introduce it each year. Anand's testimony also inspired efforts at the state level. Over the past two years, similar bills have been introduced in 25 states, and in 5 — Arkansas, Georgia, Louisiana, Minnesota and Oklahoma — they have become law. In addition, state-issued abortion-counseling materials in Alaska, South Dakota and Texas now make mention of fetal pain.

In the push to pass fetal-pain legislation, Anand's name has been invoked at every turn; he has become a favorite expert of the anti-abortion movement precisely because of his credentials. "This Oxford- and Harvard-trained neonatal pediatrician had some jarring testimony about the subject of fetal pain," announced the Republican congressman Mike Pence to the House of Representatives in 2004, "and it is

truly made more astonishing when one considers the fact that Dr. Anand is not a stereotypical Bible-thumping pro-lifer." Anand maintains that doctors performing abortions at 20 weeks or later should take steps to prevent or relieve fetal pain. But it is clear that many of the anti-abortion activists who quote him have something more sweeping in mind: changing perceptions of the fetus. In several states, for example, information about fetal pain is provided to all women seeking abortions, including those whose fetuses are so immature that there is no evidence of the existence of even a stress response. "By personifying the fetus, they're trying to steer the woman's decision away from abortion," says Elizabeth Nash, a public-policy associate at the Guttmacher Institute, a reproductive-rights group.

Another, perhaps intended, effect of fetal-pain laws may be to make abortions harder to obtain. Laura Myers, an anesthesia researcher at Children's Hospital Boston and Harvard Medical School who analyzed the Unborn Child Protection Act for the abortion-rights organization Physicians for Reproductive Choice and Health, concluded that abortion clinics do not have the equipment or expertise to supply fetal anesthesia. "The handful of centers that perform fetal surgery are the only ones with any experience delivering anesthesia directly to the fetus," Myers says. "The bill makes a promise that the medical community can't fulfill." Even these specialized centers have no experience providing fetal anesthesia during an abortion; such a procedure would be experimental and would inevitably carry risks for the woman, including infection and uncontrolled bleeding.

In his speeches about fetal pain, Senator Brownback often asks why a fetus undergoing surgery receives anesthesia but not a fetus "who is undergoing the life-terminating surgery of an abortion." Mark Rosen rejects the analogy. "Fetal surgery is a different circumstance than abortion," he says, pointing out that none of the objectives of anesthesia for fetal surgery — relaxing the uterus, for example — apply to the termination of pregnancy. That includes an objective identified just recently: preventing possible long-term damage. For the fetus that is to be aborted, there is no long term. And if there is no pain, as Rosen maintains, then there is no cause to put the woman's health at risk.

Rosen sees no contradiction in his position, only a necessary complexity. When he was in medical school, he says, he worked for a time at an abortion clinic in the morning and a fertility clinic in the afternoon — an experience that showed him "the amazing incongruities of life." In the three decades since then, he says he has come to believe that "there's a time for fetal anesthesia, and maybe there's a time not."

In their use of pain to make the fetus seem more fully human, anti-abortion forces draw on a deep tradition. Pain has long played a special role in how society determines who is like us or not like us ("us" being those with the power to make and enforce such distinctions). The capacity to feel pain has often been put forth as proof of a common humanity. Think of Shylock's monologue in "The Merchant of Venice": Are not Jews "hurt with the same weapons" as Christians, he demands. "If you prick us, do we not bleed?" Likewise, a presumed insensitivity to pain has been used to exclude some from humanity's privileges and protections. Many 19th-century doctors believed blacks were indifferent to pain and performed surgery on them without even that era's rudimentary anesthesia. Over time, the charmed circle of those considered alive to pain, and therefore fully human, has widened to include members of other religions and races, the poor, the criminal, the mentally ill — and, thanks to the work of Sunny Anand and others, the very young. Should the circle enlarge once more, to admit those not yet born? Should fetuses be added to what Martin Pernick, a historian of the use of anesthesia, has called "the great chain of feeling"? Anand maintains that they should.

For others, it's a harder call. When it comes to the way adults feel pain, science has borne out the optimistic

belief that we are all the same under the skin. As research is now revealing, the same may not be true for fetuses; even Anand calls the fetus "a unique organism." Exhibiting his flair for the startling but apt expression, Stuart Derbyshire warns against "anthropomorphizing" the fetus, investing it with human qualities it has yet to develop. To do so, he suggests, would subtract some measure of our own humanity. And to concern ourselves only with the welfare of the fetus is to neglect the humanity of the pregnant woman, Mark Rosen notes. When considering whether to provide fetal anesthesia during an abortion, he says, it's not "erring on the safe side" to endanger a woman's health in order to prevent fetal pain that may not exist.

Indeed, the question remains just how far we would take the notion that the fetus is entitled to protection from pain. Would we be willing, for example, to supply a continuous flow of drugs to a fetus that is found to have a painful medical condition? For that matter, what about the pain of being born? Two years ago, a Swiftian satire of the Unborn Child Pain Awareness Act appeared on the progressive Web site AlterNet.org. Written by Lynn Paltrow, the executive director of the National Advocates for Pregnant Women, it urged the bill's authors to extend its provisions to those fetuses "subjected to repeated, violent maternal uterine contraction and then forced through the unimaginably narrow vaginal canal."

She continued: "Imagine the pain a fetus experiences with a forceps delivery, suffering extensive bruising during and after! Shouldn't these fetuses also be entitled to their own painkillers?" And in fact, both Nicholas Fisk and Marc Van de Velde have raised the possibility of administering pain relief to fetuses undergoing difficult deliveries. Obstetricians have yet to embrace the proposal. But Sunny Anand, for one, says the idea may have merit. Though he has "misgivings about messing with a process that has worked for thousands of years," he can envision an injection of local anesthetic into the fetus's scalp where it is grasped by the forceps or vacuum device. "Let's try and work out what's best for the baby," he says.

Annie Murphy Paul is at work on a book about the lasting effects of early experience.

SUSAN B. ANTHONY LIST

Advancing Pro-life Leadership

April 18, 2012

The Honorable Trent Franks
United States House of Representatives
2435 Rayburn House Office Building
Washington, D.C. 20515

Dear Representative Franks,

On behalf of the Susan B. Anthony List and our 365,000 members across the country, we are proud to endorse your District of Columbia Pain-Capable Unborn Child Protection Act. This legislation is a top priority for the Susan B. Anthony List.

The bipartisan D.C. Pain-Capable Unborn Child Protection Act (H.R. 3803) prohibits abortions in the District of Columbia after 20 weeks gestation (about the 6-month mark), based on strong scientific evidence that an unborn child can feel pain by at least this stage of development. Article I, Section 8 of the Constitution grants Congress legislative responsibility for the District of Columbia.

There is overwhelming evidence that an unborn child has the capacity to feel pain by at least 20 weeks after fertilization, and often even sooner. Yet in our nation's capital, abortions can be performed for any reason before birth, allowing some abortionists to travel to the D.C. area from states where late-term abortions are restricted.

Right now, babies are aborted by the D&E method in the District of Columbia, a method in which their arms and legs are ripped apart by brute manual force. One abortionist even reportedly takes a giant needle and stabs the unborn child in the heart. This is an unacceptable tragedy, and the D.C. Pain-Capable Unborn Child Protection Act will put an end to this injustice.

Six states (Kansas, Nebraska, Oklahoma, Idaho, Alabama, and Arizona) have already passed similar legislation, and it is time that the Congress uses its legislative authority to do the same in our nation's capital.

The SBA List is proud to endorse this bipartisan legislation that protects women and pain-capable unborn children from the horrors of late-term abortion. We sincerely thank you for introducing this common sense, live-saving legislation.

For Life,

Marjorie Dannenfelser
President

Marilyn Musgrave
Vice President of Government Affairs

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Material submitted by the Honorable Jerrold Nadler, a Representative in Congress from the State of New York, and Ranking Member, Subcommittee on the Constitution

Written Statement of the American Civil Liberties Union

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Submitted to the U.S. House of Representatives
Committee on the Judiciary
Subcommittee on the Constitution

Hearing on: H.R. 3803, the "District of Columbia Pain-Capable Unborn Child Protection Act"

Held May 17, 2012

On behalf of the American Civil Liberties Union (ACLU), a non-partisan organization with more than a half million members, countless additional activists and supporters, and 53 affiliates nationwide, and the American Civil Liberties Union of the Nation's Capital, with more than 5,000 members in the District of Columbia, both dedicated to protecting the principles of freedom and equality set forth in the Constitution and in our nation's civil rights laws, we thank you for giving us the opportunity to submit this statement for the record on the so-called District of Columbia Pain-Capable Unborn Child Protection Act, H.R. 3803, which would ban abortions in the District of Columbia at 20 weeks.

The ACLU has a long history of defending reproductive freedom. The ACLU has participated in nearly every critical case concerning reproductive rights to reach the Supreme Court, and we routinely advocate in Congress and state legislatures for policies that promote access to reproductive health care. We oppose H.R. 3803 because it is unconstitutional and interferes in a woman's most personal, private medical decisions, and unduly targets the residents of the District of Columbia.

Every pregnancy is different. For many women and families, it is a joyous event. However, none of us can presume to know what complications may arise during the pregnancy, or all the circumstances surrounding a personal, medical decision to have an abortion. This is an inherently private decision that must be made by a woman and her family, not the government, and the United States Supreme Court has long recognized as much. In *Roe v. Wade*, the Court specifically held that: (1) a state may never ban abortion prior to fetal viability; and (2) a state may only ban abortion after viability if there are adequate exceptions to protect a woman's life *and* health.[1] These principles have been repeatedly reaffirmed for more than three decades,[2] as well they should. A woman should not be denied basic health care or the ability to make the best decision for her circumstances just because some disagree with her decision. H.R. 3803 flouts these basic rules.

In conflict with law, in disregard of medical science, and for reasons unrelated to viability, H.R. 3803 unilaterally takes away a woman's decision-making ability before viability and fails to provide even adequate protection for a woman's health. Banning abortions starting at 20 weeks – which is a pre-viability stage of pregnancy – directly contradicts longstanding

[1] 410 U.S. 113, 163-64 (1973).

[2] Gonzales v. Carhart, 550 U.S. 124, 145 (2007) ("It must be stated at the outset and with clarity that *Roe's* essential holding, the holding we reaffirm, has three parts. First is a recognition of the right of a woman to choose to have an abortion before viability and obtain it without undue interference from the State. Before viability, the State's interests are not strong enough to support a prohibition of abortion or the imposition of a substantial obstacle to the woman's effective right to elect the procedure."). *See also* Planned Parenthood v. Casey, 505 U.S. 833, 871 (1992) (plurality opinion) ("The woman's right to terminate her pregnancy before viability is the most central principle of *Roe v. Wade*. It is a rule of law and a component of liberty we cannot renounce.").

precedent holding that a woman should "be free from unwarranted governmental intrusion" when deciding whether to continue or terminate a pre-viability pregnancy.[3]

The Supreme Court has long been clear that a legislature cannot declare any one element – "be it weeks of gestation or fetal weight or any other single factor – as the determinant" of viability."[4] Similarly here, the government cannot draw a line based on any single factor to prohibit abortions. Thus, a 20-week ban on abortions, no matter the justification, is by definition unconstitutional. In fact, a similar 20-week provision enacted by the Utah legislature has already been struck down as unconstitutional by the United States Court of Appeals for the 10th Circuit because it "unduly burden[ed] a woman's right to choose to abort a nonviable fetus."[5]

Moreover, H.R. 3803 provides only a single, exceedingly narrow, exception to its ban: where the abortion is "necessary to save the life of a pregnant woman whose life is endangered by a physical disorder, physical illness, or physical injury."[6] Put differently, H.R. 3803 bans abortions necessary to protect a woman's health. Many things can go wrong during a pregnancy; a woman's health could be at risk in ways that we cannot predict. Women may suffer blindness, kidney failure, or permanent infertility because they were denied the care they need by this bill. H.R. 3803 would force a woman and her doctor to wait until her condition was terminal to finally act to protect her health, but by then it may be too late. Such a restriction is as unconstitutional as it is cruel. It is longstanding precedent that restrictions on abortion post-viability must have an exception to preserve a woman's health.[7] This is all the more so true here where the ban impermissibly applies pre-viability.

The disregard for women's health displayed by H.R. 3803 knows almost no limit. Even when a woman qualifies for the narrow life exception – that is, when her life is literally in peril – H.R. 3803 goes out of its way to further tie her doctor's hands. The bill dictates how the pregnancy termination must be performed, even if such a method will put a woman's health at greater risk.[8] In other words, this bill disallows a doctor from choosing the method of abortion that will best protect a woman's health.

In addition to ignoring – indeed, sacrificing – women's health, H.R. 3803 fails to take into consideration the fatal fetal conditions that develop or are detected in mid or later pregnancy. Consider the turmoil that Danielle Deaver suffered when her water broke months

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[3] *Casey*, 505 U.S. at 851.
[4] Colautti v. Franklin, 439 U.S. 379, 388-89 (1979).
[5] Jane L. v. Bangerter, 102 F.3d 1112, 1118 (10th Cir. 1996).
[6] District of Columbia Pain-Capable Unborn Child Protection Act, H.R. 3803, 112th Cong. § 3 (2012).
[7] *Casey*, 505 U.S. at 879 (a post-viability ban must make an exception where an abortion is "necessary, in appropriate medical judgment, for the preservation of the life *or health*" of the woman) (emphasis added); *see also Roe*, 410 U.S. at 164-65.
[8] There are only two narrow exceptions to this provision: when such a method would pose greater risk of death or the substantial and irreversible physical impairment of a major bodily function.

early at 22 weeks. She sped to the hospital, only to be told that her fetus had no chance of survival – her lungs would never develop; she would never be able to breathe. Danielle and her husband made the best decision for their family – to end the pregnancy and their own suffering, and spare their baby any pain. Tragically for Danielle, the state of Nebraska had already enacted an abortion ban similar to H.R. 3803, and her doctors were therefore not able to give her the care she needed and so desperately sought. She was forced to sit and wait for 10 days until her body finally expelled the pregnancy. In Danielle's words: "There are no words for how awful the 10 days were from the moment my water broke to the day my daughter died. There are no words for the heartbreak that cut deeper every time she moved inside of me for those 10 days."[9]

Last, H.R. 3803 impinges on the autonomy of the District of Columbia. This ban tramples on the core concept of home rule. Although our Constitution gave Congress the authority to establish a federal district, the District of Columbia, Senators and Representatives holding widely divergent political views, finally recognized in 1973 that the citizens of the District of Columbia had been denied the most basic privilege enjoyed by all other Americans – the right to elect those men and women who would control their local governments. They enacted the Home Rule Act to "grant to the inhabitants of the District of Columbia powers of local self-government...and relieve Congress of the burden of legislating upon essentially local District matters."[10]

The 20 week ban is antithetical to the spirit of the Home Rule Act. It disenfranchises and marginalizes the District's leaders and residents. Through this provision, non-resident Members of Congress impose their own ideology, morality or belief upon the District's residents and disregard the needs or wishes of the broader community or those directly impacted. Members of the House who seek to impose this abortion ban and negate the will of the District's residents are not accountable to the people of the District.

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We may not all agree on abortion, but we can all agree that it is important to support a woman's health and well-being. This bill is should be rejected, not just because it is unconstitutional, but because it puts politics above a woman's health. We urge the members of the Subcommittee to oppose this dangerous bill.

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[9] *See* Mathew Hendley, *Nebraska Woman Lets Jan Brewer Know Proposed Abortion Bill Actually Affects People*, PHOENIX NEWS TIMES, April 5, 2012, *available at* http://blogs.phoenixnewtimes.com/valleyfever/2012/04/nebraska_woman_lets_jan_brewer_1.php.

[10] District of Columbia Home Rule Act, Pub. L. No. 93-198, 87 Stat. 774,777 (1973).

May 17, 2012

Dear Members of the House Judiciary Subcommittee on the Constitution:

I am writing regarding the testimony that has been submitted in support of H.R. 3803, the so-called District of Columbia Pain-Capable Unborn Child Protection Act. As an obstetrician-gynecologist with more than 20 years of experience providing both obstetric and complex abortion care, I wish to set the record straight.

I direct Northwestern University's Center for Family Planning & Contraception as well as its academic Section of Family Planning. The medical center where I work performs nearly 13,000 deliveries annually. Most patients are healthy women having healthy babies, but I am frequently asked to provide abortions for women confronting severely troubled pregnancies or their own life-endangering health issues. Physicians who provide health care to women cannot choose to ignore the more tragic consequences of human pregnancy—and neither should Congress.

The witnesses inaccurately claim that the gestational limits contained in the bill, defined as a "probable post-fertilization age . . . of the unborn child [of] 20 weeks or greater" are not vague because post-fertilization dating is as valid as LMP dating. This is incorrect. The common reference point used by all clinical obstetric providers for dating a pregnancy—including normative data for most obstetric ultrasound—is dating derived from or compared with a woman's last menstruation period (LMP). This is because there is a relative amount of certainty as to when a woman had her last period. Fertilization generally takes place between 2 - 4 weeks after the last menstruation period. Although in an "ideal" cycle the assumption would be that the fertilization occurred 2 weeks after LMP, the reality is that timing of ovulation can vary between women and between cycles in an individual woman.

Because H.R. 3803 bases its language on post-fertilization dating, which physicians cannot always definitively establish, physicians will be unable to determine whether or not they are in violation of the law when treating women with many pregnancies earlier than 22 weeks LMP. This is particularly true in those patients whose last menstrual period is uncertain or unverified by early ultrasound. Even the witnesses establish post-fertilization dating in their testimony by referencing LMP—they simply add two weeks to the LMP and state that it is the post-fertilization date. Their assumption that "LMP + 2" is an accurate substitute for the post-fertilization date is completely unfounded. Why would Congress accept language so clearly unacceptable to the majority of obstetric providers throughout our nation?

Unfortunately, the language Congress uses has real implications for the health and well being of my patients. The witnesses claim that abortion care is never necessary to save a woman's life and cannot be instituted sooner than 36 hours. These claims are also untrue.

The following portraits of the women I see illustrate just a few of the circumstances where abortion saves women's lives. In many of the cases we have saved women's lives through immediate abortion with little or no cervical preparation:

- One of my own obstetric patients carrying a desired pregnancy recently experienced rupture of the amniotic sac at 20 weeks gestation. The patient had a complete placenta previa, a condition where the afterbirth covers the opening to the uterus. Although the patient hoped the pregnancy might continue, she began contracting and suddenly hemorrhaged, losing nearly a liter of blood into her bed in a single gush. Since the patient's cervix had already dilated sufficiently, I was able to take this patient to the operating room and perform an immediate dilation and evacuation procedure without need for pre-operative cervical ripening. Had we not quickly intervened to terminate the pregnancy, she would have bled to death, just as women do in countries with limited access to obstetric services. Had we not performed an immediate dilation and evacuation procedure, the patient would have required a procedure resembling a cesarean section—but associated with far greater blood loss than a cesarean, greater maternal risk than most cesareans and posing far greater risk to the patient's subsequent pregnancies than most cesarean sections. This abortion saved the patient's life, protected her health and protected the health of future pregnancies. She required no pre-operative cervical preparation.

- My service frequently receives referrals from Northwestern's Division of Maternal Fetal Medicine and other high risk pregnancy services throughout the Chicago area. One of the more frequent reasons for referral is preterm rupture of membranes with chorioamnionitis, an intrauterine infection which can develop at any time during pregnancy. Since antibiotics will not sufficiently penetrate the endometrial cavity containing the baby, the treatment for this condition is to evacuate the uterus. If the infection occurs at term, we deliver the baby. If the condition occurs before 24 weeks, we must abort the pregnancy lest the patient become septic and die. Over my years of practice, I have had many patients who would have died without access to abortion in this situation. In many septic patients, the cervix begins to ripen permitting us to substantially shorten traditional cervical ripening regimens or perform mechanical dilation with immediate uterine evacuation. I cannot remember a case such as this that ever required 36 hours of cervical preparation.

- My service often receives consults regarding patients with serious medical issues complicating pregnancy. We recently had a 44-year-old patient whose pregnancy had been complicated by a variety of non-specific symptoms. A CT scan obtained at 23 weeks gestation revealed that the patient had lung cancer that had metastasized to her brain, liver, and other organs. Her family confronted the difficult choice of terminating a desired pregnancy or continuing the pregnancy knowing that the physiologic burden of pregnancy and cancer might worsen her already poor prognosis. The family chose to proceed with pregnancy termination.

- My service often receives referrals regarding unusual obstetric conditions because we work at a tertiary care center. One complex condition referred to my service involved a patient who had a twin gestation in which one of the embryos was a molar pregnancy. Molar pregnancy is an abnormal pregnancy in which the embryo fails to develop—or develops partially—and the placenta develops into grape like tissue clusters. The abnormal placenta of molar gestation expands the uterine cavity and often causes severe hemorrhage. Patients are also more likely to develop a number of other medical problems during their pregnancy including intractable nausea and vomiting and early onset hypertensive disorders. Longer term, molar gestation places the patient at higher risk of developing choriocarcinoma, a cancer in which placenta-like material spreads throughout the body. Most molar gestations involve no embryo, but this patient had one normal twin and one molar gestation. Although she was only 22 weeks gestation, her uterus already approximated the size of a term pregnancy containing enough grape like clusters of placenta to fill a milk crate. We admitted the patient to the intensive care unit, obtained 10 units of blood in case severe bleeding occurred, and successfully terminated the pregnancy. By intervening when we did, we preserved the patient's life, her health, and her ability to have children in the future.

- My service sometimes sees patients who have received organ transplants or are awaiting transplants. I remember one woman in her early twenties who had end stage alcoholic cirrhosis of the liver. She had stopped using alcohol and successfully balanced school, work, and frequent hospitalizations to deal with her severe liver disease and related disorders. While awaiting a transplant, she conceived. She decided to terminate the pregnancy rather than accept the risks to her life and health posed by continued gestation. We have cared for other patients who chose to terminate while awaiting transplant or after undergoing transplant of heart, liver, and other organs. Although some of these patients might manage to continue pregnancies to term, each patient's circumstance is highly variable with unpredictable risk to life and health.

- A colleague on my team recently took care of another patient with leukemia. We have had many during my 17 years at Northwestern. Several years ago, we had three patients with leukemia requiring pregnancy terminations at approximately the same time. Because leukemia causes abnormal blood cells, patients with leukemia confront increased risk of both bleeding and infection. Pregnancy compounds these risks, particularly if they need to receive ongoing chemotherapy during the pregnancy.

- My service frequently sees patients with early pre-eclampsia, often referred to by the term "toxemia". Pre-eclampsia usually complicates later gestation, but occasionally complicates pregnancy as early as 18 to 20 weeks, well before the fetus is viable. The only treatment for severe pre-eclampsia is delivery. Otherwise, the condition will worsen, exposing the mother to kidney failure, liver failure, stroke and death. One Christmas morning I had to leave my own family so that I could provide a pregnancy termination for a remarkably sick, pre-eclamptic teenager.

Patients like those described above rarely knew that pregnancy could jeopardize their lives and health. Some opposed "abortion", even while they themselves were undergoing an abortion. Like most tertiary obstetric centers, we receive referrals of such patients from within our own system and throughout our metropolitan area. Some of the referrals come from providers or sectarian institutions that ostensibly oppose abortion, but rely upon us as the "safety valve" to assure that patients get the care they need and deserve.

We usually manage to intervene before a risk to health becomes a risk of life, but we do so because the law currently embraces patient and provider autonomy. What will obstetricians do when the law criminalizes interventions needed to save the lives of our daughters, wives, and mothers?

I hope our elected representatives will allow those of us who experience these circumstances on a regular basis to set the record straight—and prevent the passage of legislation that would harm women, families, and those who care for them.

Cassing Hammond, MD

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Current Member and Immediate Past Chair
National Abortion Federation Board of Directors

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NAF is the professional association of abortion providers in North America. Our members include private and non-profit clinics, Planned Parenthood affiliates, women's health centers, physicians' offices, and hospitals who together care for more than half the women who choose abortion in the U.S. and Canada each year. Our members also include public hospitals and both public and private clinics in Mexico City.

For more information, please contact NAF's public policy department at 202-667-5881.

February 24, 2010

Judiciary Committee
Nebraska Legislature
Room 1103, State Capitol
Lincoln, Nebraska 68509

Re: Constitutionality of LB 1103

Dear Judiciary Committee Members,

This letter explains why, based on the most current precedent of the United States Supreme Court, it is my opinion that LB 1103, the Abortion Pain Prevention Act, is unconstitutional both because it impermissibly bans abortions prior to fetal viability and because it fails to protect women's health adequately.*

I. A Woman's Constitutional Right to Choose

For more than thirty-five years, the U.S. Supreme Court has recognized that the constitutional rights to liberty and privacy extend to the decision of a woman to terminate her pregnancy. The Court first reached this conclusion in the landmark decision of *Roe v. Wade*, 410 U.S. 113 (1973), where the Court specifically held that: (1) a state may not ban abortion prior to fetal viability; and (2) a state may ban abortion after viability so long as there are exceptions to protect the woman's health and life. *Id.* at 163-64 ("If the State is interested in protecting fetal life after viability, it may go so far as to proscribe abortion during that period, except when it is necessary to preserve the life or health of the mother."). The Court explained in *Roe* that viability was that point in pregnancy when the fetus is "potentially able to live outside the mother's womb, albeit with artificial aid." *Id.* at 160.

In *Planned Parenthood of Southeastern Pennsylvania v. Casey*, 505 U.S. 833 (1992), the Supreme Court reaffirmed these central tenets of *Roe*. *Id.* at 878-79. The plurality opinion, joined by Justice Anthony Kennedy, specifically held that "viability marks the earliest point at which the State's interest in fetal life is constitutionally adequate to justify a legislative ban on nontherapeutic abortions." *Id.* at 860; *see also id.* at 870 ("We conclude the line should be drawn

* I am the Douglas B. Maggs Professor Emeritus at Duke Law School, where I am now on leave, and a Visiting Professor of Law at Harvard University. I am also currently practicing law in Washington, DC. From 1993 to 1996, I served as Assistant Attorney General and head of the Office of Legal Counsel (OLC) at the United States Department of Justice. During the 1996-1997 term of the U.S. Supreme Court, I was acting Solicitor General. I continue to practice and argue cases before the U.S. Supreme Court. For example, in the 2007-2008 term of the Court, I argued *Morgan Stanley v. Public Utility District*, *Exxon v. Baker*, and *Heller v. District of Columbia*. In all of these capacities, it has been my job to consider and argue the constitutionality of a wide variety of issues that impact our national policy. I have also published articles on constitutional issues for scholarly journals including the *Harvard Law Review*, the *Yale Law Journal*, and the *Duke Law Journal*, and have written for the *New York Times*, the *Washington Post*, *Newsweek*, the *New Republic*, and the *London Times*. I have testified more than twenty-five times before committees of the United States Congress, many times on constitutional issues.

at viability, so that before that time the woman has a right to choose to terminate her pregnancy.").

The constitutional protection for a woman's decision to end her pregnancy derives from the Due Process Clause of the Fourteenth Amendment to the Constitution. The Supreme Court has consistently ruled that the Due Process Clause protects the right to "substantive liberties," including the right to "a realm of personal liberty which the government may not enter." *Casey*, 505 U.S. at 847. Because of the inherently private nature of the "decision whether to bear or beget a child," the Supreme Court has recognized that individuals have the right to "be free from unwarranted governmental intrusion" when deciding whether to continue or terminate a pre-viability pregnancy. *Casey*, 505 U.S. at 851. This right to choose a pre-viability abortion without undue interference from the government applies *regardless of why* the woman has chosen to end her pregnancy and regardless of why the state might seek to restrict that choice.

LB 1103 violates these principles, as explained in more detail below, both because it bans *pre-viability* abortions and because even if it applied only *post-viability*, the narrow exceptions to the ban fail to adequately protect a woman's health.

II. LB 1103 is Unconstitutional Because It Bans Pre-Viability Abortions

In the years following *Roe*, the Supreme Court had numerous opportunities to reconsider its decision both that states may not ban abortion prior to viability and what viability in this context means. It has never wavered. For example, three years after *Roe*, in *Planned Parenthood of Central Missouri v. Danforth*, 428 U.S. 52 (1976), the Court upheld a definition of "viability" in a Missouri statute because it allowed for the necessary "flexibility of the term." *Id.* at 64. The *Danforth* Court specifically rejected the "contention that a specified number of weeks in pregnancy must be fixed by statute as the point of viability." *Id.* at 65. The Court explained:

> [I]t is not the proper function of the legislature or the courts to place viability, which essentially is a medical concept, at a specific point in the gestation period. The time when viability is achieved may vary with each pregnancy, and the determination of whether a particular fetus is viable is, and must be, a matter for the judgment of the responsible attending physician.

Id. at 64.

Three years after *Danforth*, in *Colautti v. Franklin*, 439 U.S. 379 (1979), the Court considered a Pennsylvania law regulating post-viability abortion and reaffirmed both that viability was the earliest point at which the state could ban abortion and that the determination of viability must not be fixed in weeks, but rather is a matter to be left to the physician's judgment. The Court explained:

> [T]his Court has stressed viability, has declared its determination to be a matter for medical judgment, and has recognized that differing legal consequences ensue upon the near and far sides of that point in the human gestation period. We

> reaffirm these principles. Viability is reached when, in the judgment of the attending physician on the particular facts of the case before him, there is a reasonable likelihood of the fetus' sustained survival outside the womb, with or without artificial support. Because this point may differ with each pregnancy, neither the legislature nor the courts may proclaim one of the elements entering into the ascertainment of viability – be it weeks of gestation or fetal weight or any other single factor – as the determinant of when the State has a compelling interest in the life or health of the fetus. Viability is the critical point.

Id. at 388-89.

A decade later, in *Webster v. Reproductive Health Services*, 492 U.S. 490 (1989), the Court reiterated its holdings in *Danforth* and *Colautti* that the determination of viability is a matter for the judgment of the attending physician. *See id.* at 516-17 (plurality); *id.* at 526-27 (O'Connor, J., concurring); *id.* at 545 n.6 (Blackmun, J., joined by Brennan, J., and Marshall, J., concurring and dissenting). And, as noted above, in *Casey*, the Court once again concluded that "the line should be drawn at viability, so that before that time the woman has a right to choose to terminate her pregnancy." 505 U.S. at 870.

LB 1103 is in clear violation of these principles. It does not draw its line at viability – it draws the line at 20 weeks after fertilization. As explained above, the Supreme Court has specifically – and repeatedly – rejected the notion that abortion may be banned at a specific point in pregnancy.[1] Instead, it has always held that viability is the earliest point at which a ban may apply – and that the determination of when viability is reached must be left to the physician.

Of perhaps particular note, Utah enacted an abortion statute similar to LB 1103 in 1991. *See Jane L. v. Bangerter*, 102 F.3d 1112, 1114 (10th Cir. 1996) (considering statute that banned most abortions after "20 weeks gestational age, measured from the date of conception"). The Tenth Circuit struck down the law, ruling that Utah's attempt to legislate the viability determination, rather than permit physicians to exercise their judgment about viability, "is directly contrary to the Supreme Court authority." *Id.* at 1115. The Tenth Circuit explained:

> [T]he State made a deliberate decision to disregard controlling Supreme Court precedent set out in *Roe*, *Danforth*, *Colautti*, and *Webster*, and to ignore the Supreme Court's repeated directive that viability is a matter for an attending physician to determine. In our view, the State's determination to define viability in a manner specifically and repeatedly condemned by the Court evinces an intent to prevent a woman from exercising her right to choose [a previability] abortion . . . and it therefore imposes an unconstitutional undue burden on her right to choose.

Id. at 1116-17 (footnote omitted).

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[1] The point of viability cannot be legislated with a number of weeks because it may differ with each pregnancy. Nonetheless, a fetus is not "generally understood to have achieved viability – meaning that there exists a realistic potential for long-term survival outside the uterus [until] twenty-four weeks lmp or later." *Planned Parenthood Fed'n of Am. v. Gonzales*, 435 F.3d 1163, 1166 n.1 (9th Cir. 2006).

A court looking at LB 1103 would have no choice but to reach the same conclusion as the Tenth Circuit did – LB 1103 is in deliberate disregard of the Supreme Court's longstanding precedent, and therefore, unconstitutional.

III. LB 1103 is Unconstitutional Because it Threatens Women's Health

The only exception to LB 1103's prohibition on performing an abortion after 20 weeks from fertilization is if the woman "has a condition which so complicates her medical condition as to necessitate the abortion . . . to avert her death or to avert serious risk of substantial and irreversible physical impairment of a major bodily function."

As is explained above, the Supreme Court has long held that after viability, a state may prohibit abortion, but that prohibition must make exception for where abortion "is necessary, in appropriate medical judgment, for the preservation of the life *or health*" of the woman. *Casey*, 505 U.S. at 879 (emphasis added); *see also Roe*, 410 U.S. at 165.

While the Supreme Court has said that language similar to LB 1103's exception is an adequate *medical emergency* exception for restrictions that delay abortions such as a 24-hour waiting period or a parental consent requirement, *Casey*, 505 U.S. at 880, the Court has never upheld similarly narrow language as an adequate health exception for a complete abortion *ban* such as LB 1103. Furthermore, the medical emergency exception that the Supreme Court upheld in *Casey* was not limited to "physical" health as LB 1103 is.

In fact, the Court has rejected the notion that the protection afforded to women's health by an abortion restriction may be so limited. *See Doe v. Bolton*, 410 U.S. 179, 192 (1973) ("[T]he medical judgment may be exercised in the light of all factors – physical, emotional, psychological, familial, and the woman's age – relevant to the well-being of the patient. All these factors may relate to health."); *cf. Casey*, 505 U.S. at 882 ("It cannot be questioned that psychological well-being is a facet of health."); *Thornburgh v. ACOG*, 476 U.S. 747, 768-69 (1986) (invalidating *post-viability* abortion restriction because it placed pregnant women at medical risk by failing to require maternal health to be the "physician's paramount consideration"); *Women's Med. Prof'l Corp. v. Voinovich*, 911 F. Supp. 1051, 1080-81 (S.D. Ohio 1995) (holding post-viability abortion restriction unconstitutional because "a state may not constitutionally limit the provision of abortions only to those situations in which a pregnant woman's *physical health* is threatened, because this impermissibly limits the physician's discretion to determine what measures are necessary to preserve her health") (emphasis added), *aff'd on other grounds*, 130 F.3d 187 (6th Cir. 1997).

Because LB 1103 allows abortions only if necessary "to avert serious risk of substantial and irreversible physical impairment of a major bodily function," it would be unconstitutional even if it applied only *post-viability* (which it does not), because it does not allow all abortions that may be necessary for the preservation of the health of the woman.

IV. The Supreme Court's Decision in *Gonzales v. Carhart* Does Not Alter this Result

I understand that some proponents of LB 1103 believe that the case law I have discussed above is in doubt following the Supreme Court's 2007 decision in *Gonzales v. Carhart*, 550 U.S. 124 (2007) ("*Carhart II*"), which upheld the federal ban on "partial-birth abortion." For the following reasons, I do not believe that to be so.

First and foremost, the Court in *Carhart II* did not overrule any of its previous precedent. Indeed, the Court went to great lengths to explain why its decision to uphold the federal ban was fully consistent with both *Casey* and its earlier ruling in *Stenberg v. Carhart*, 530 U.S. 914 (2000) ("*Carhart I*"), which struck down a similar Nebraska law. *See, e.g.*, *Carhart II*, 550 U.S. at 146 (stating that the Court was "apply[ing]" the standard set forth in *Casey*); *id.* at 151-52 (differentiating the federal ban from the Nebraska ban upheld in *Carhart I*).

Moreover, while it is true that the federal partial-birth abortion ban does not contain an exception to protect women's health, the Court was clear in *Carhart II* that it could uphold that law only because it reached just *one method* of abortion. Central to the Court's holding was the fact that other methods of abortion remained available in all instances, and especially if a woman needed an abortion to protect her health. The Court explained that Congress could ban one method of abortion without a health exception "given the availability of other abortion procedures that are considered to be safe alternatives." 550 U.S. at 167; *see also id.* at 164 ("Alternatives are available to the prohibited procedure."); *id.* at 165 ("Here the Act allows, among other means, a commonly used and generally accepted method, so it does not construct a substantial obstacle to the abortion right.").

In contrast, LB 1103 bans *all* abortions after 20 weeks after fertilization – not just one method. Therefore, if a woman needs an abortion to protect her health that does not meet the bill's narrow exception, she would not have a safe alternative to end her pregnancy, as Supreme Court precedent – including *Carhart II* – has required for more than thirty-five years.

The *Carhart II* Court's treatment of the interests that a state may assert in order to justify an abortion restriction has garnered much debate. However, that discussion does not alter LB 1103's unconstitutionality. There is nothing in *Carhart II* that suggests that a state can ban all abortions at any point prior to viability – regardless of the interest it asserts in doing so. Nor did the Court hold that a state could put forth an interest that would overcome the constitutional protection required for women's health.[2]

Finally, contrary to some suggestions, nothing in the recent changes to the composition of the Court alters my conclusion. The decisions in both *Carhart I* and *Carhart II* were decided by a vote of 5 to 4. In both cases, Justice Kennedy voted to uphold the ban on partial-birth abortion. Therefore, *Carhart II* was consistent with his prior vote dissenting in *Carhart I*. However, as I mentioned earlier, Justice Kennedy is also one of the members of the Court who joined the plurality opinion in *Casey*, which LB 1103 clearly violates. Therefore, in order for the U.S. Supreme Court to uphold LB 1103, Justice Kennedy would have to disavow his prior opinion in

[2] While there is nothing contrary to these principles in Justice Ginsburg's dissent in *Carhart II*, the analysis would be the same even if there were – it is axiomatic that dissenting opinions have no precedential force.

Casey, something that I believe that he is unlikely to do, especially given the strong endorsement of *Casey* in the majority opinion in *Lawrence v. Texas*, 539 U.S. 558 (2003) which Justice Kennedy authored. *See* 539 U.S. at 573-74.

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For all of these reasons, it is my opinion that the most recent and controlling precedent of the United States Supreme Court leads to only one conclusion – LB 1103 is an unconstitutional restriction on a woman's right to choose, and would be found so by the federal courts.

Sincerely yours,

Walter Dellinger

Walter Dellinger
Former Acting Solicitor General

CENTER FOR REPRODUCTIVE RIGHTS

Testimony of the Center for Reproductive Rights

Hearing: H.R. 3803, "District of Columbia Pain-Capable Unborn Child Protection Act"

May 17, 2012

The Center for Reproductive Rights respectfully submits the following testimony to the House Judiciary Committee, Subcommittee on the Constitution. Since 1992, the Center for Reproductive Rights has worked toward the time when the promise of reproductive freedom is enshrined in law in the United States and throughout the world. We envision a world in which every woman is free to decide whether and when to have children; every woman has access to the best reproductive healthcare available; and every woman can make medical decisions without coercion or discrimination. More simply put, we envision a world in which every woman participates with full dignity as an equal member of society.

We urge this Subcommittee to vote against H.R. 3803, the so-called "District of Columbia Pain-Capable Unborn Child Protection Act," as it:

I. Violates settled law that bans on abortion before viability and bans that do not protect women's health violate the Constitution;

II. Tramples on the autonomy of the residents and elected representatives of the District of Columbia;

III. Would harshly penalize compassionate medical providers and subject them to a threat of litigation;

IV. Would ban virtually all abortions after 20 weeks gestation, regardless of the woman's circumstances; and

V. Is based on unproven claims that have no basis in medical science.

H.R. 3803 violates women's basic human rights by taking a deeply personal, medical decision that should be made by a woman and her doctor and substituting the judgment of politicians. Every pregnancy is different, and no one can presume to know all of the circumstances surrounding the decision to have an abortion. It is cruel and degrading to women to give to the

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government the power to make one sweeping rule that applies to all situations without regard for individual circumstances. H.R. 3803 should be rejected as an attack on reproductive rights and the District of Columbia's right to self-government.

I. THE ACT IS BLATANTLY UNCONSTITUTIONAL

The United States Constitution prohibits the government from enacting a law that bans abortion prior to the point in pregnancy when a fetus is viable, and prohibits the government from drawing a line at a particular gestational age to establish when viability begins.[1] Although the point of viability, "meaning [the] realistic potential for long-term survival outside the uterus," differs with each pregnancy, a fetus is not "generally understood to have achieved viability . . . [until] twenty-four weeks lmp[2] or later."[3] The twenty week line in H.R. 3803 is at least two full weeks before the "generally understood" advent of viability. By completely banning some pre-viability abortions, H.R. 3803 directly conflicts with U.S. Supreme Court precedent on abortion.[4]

As the Supreme Court has said repeatedly, "viability marks the earliest point at which the State's interest in fetal life is constitutionally adequate to justify a legislative ban on nontherapeutic abortions."[5] The Supreme Court has never wavered from this position, despite numerous opportunities to do so.

The Court has emphasized that "viability" is necessarily a "flexib[le] . . . term," and that the government *cannot* "place viability, which essentially is a medical concept, at a specific point in

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[1] *See Roe v. Wade*, 410 U.S. 113, 163-64 (1973). In *Gonzales v. Carhart* ("*Carhart II*"), 550 U.S. 124 (2007), the most recent Supreme Court case on abortion, the law at issue did not ban abortions in general or abortions at any particular point in pregnancy. Rather, it banned only *one abortion procedure*. Although the Supreme Court upheld that law, the Court emphasized that safe alternative abortion procedures were available and explained that its decision was fully consistent with past precedent. *See, e.g., id.* at 146 (stating that the decision is guided by the principle, *inter alia*, that "[b]efore viability, a State 'may not prohibit any woman from making the ultimate decision to terminate her pregnancy,'" quoting *Casey*).

[2] Sometimes gestational age is measured using the date of the pregnant woman's last menstrual period (lmp), which would typically be two weeks prior to fertilization. H.R. 3803 uses fertilization as the beginning of gestational dating: 20 weeks post-fertilization is 22 weeks lmp.

[3] *Planned Parenthood Fed'n of America v. Gonzales*, 435 F.3d 1163, 1166 n.1 (9th Cir. 2006), *rev'd on other grounds*, *Gonzales v. Carhart*, 550 U.S. 124 (2007).

[4] *See also* letter from Walter Dellinger, former Solicitor General of the United States, outlining why a state law with similar provisions to H.R. 3803 is unconstitutional (attached).

[5] *Planned Parenthood of S.E. Pa. v. Casey*, 505 U.S. 833, 860 (1992); *see also id.* at 870 ("We conclude the line should be drawn at viability, so that before that time the woman has a right to choose to terminate her pregnancy."); *id.* at 879.

the gestation period."[6] Moreover, because "[t]he time when viability is achieved may vary with each pregnancy,"[7] the Court also has insisted that the determination of viability must be left to the physician's judgment.[8] H.R. 3803 directly contradicts these important constitutional principles.

Notably, in 1996 the United States Court of Appeals for the Tenth Circuit struck down a Utah statute that, like H.R. 3803, banned abortion after twenty weeks gestation. *Jane L. v. Bangerter*, 102 F.3d 1112, 1114 (10th Cir. 1996). That court held that Utah's attempt to legislate the viability determination was "directly contrary to the Supreme Court authority," and found that the state's "deliberate decision to disregard controlling Supreme Court precedent set out in *Roe*, *Danforth*, *Colautti*, and *Webster*, and to ignore the Supreme Court's repeated directive that viability is a matter for an attending physician to determine" showed that the state intended "to prevent a woman from exercising her right to choose [a previability] abortion" and imposed "an unconstitutional undue burden on her right to choose." *Id.* at 1115-17 (footnote omitted).

H.R. 3803 is constitutionally infirm on other grounds as well, as it would allow a post-20 week abortion in the District only if a woman's *life* is in danger. The bill would prohibit physicians from providing such abortions to women whose *health* is placed at risk by the pregnancy -- leaving women with illness such as cancer, heart disease, or myriad other serious conditions without the ability to get essential medical care. Since recognizing the constitutional right to choose an abortion, the Supreme Court has consistently held that even though a state may ban abortion *after* viability, any such ban must make an exception when an abortion "is necessary, in appropriate medical judgment, for the preservation of the life *or health*" of the woman.[9] Because

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[6] *Planned Parenthood of Cent. Mo. v. Danforth*, 428 U.S. 52, 64 (1976).

[7] *Id.*

[8] *Colautti v. Franklin*, 439 U.S. 379 (1979). "Viability is reached when, in the judgment of the attending physician on the particular facts of the case before him, there is a reasonable likelihood of the fetus' sustained survival outside the womb, with or without artificial support. Because this point may differ with each pregnancy, neither the legislature nor the courts may proclaim one of the elements entering into the ascertainment of viability – be it weeks of gestation or fetal weight or any other single factor – as the determinant of when the State has a compelling interest in the life or health of the fetus. Viability is the critical point." *Id.* at 388-89; *see also Casey*, 505 U.S. at 870 (holding again that "the line should be drawn at viability, so that before that time the woman has a right to choose to terminate her pregnancy"); *Webster v. Reprod. Health Servs.*, 492 U.S. 490 (1989) (holding that the determination of viability is a matter for the judgment of the attending physician); *see id.* at 516-17 (plurality opinion); *id.* at 526-27 (O'Connor, J., concurring); *id.* at 545 n.6 (Blackmun, J., joined by Brennan, J., and Marshall, J., concurring and dissenting).

[9] *Roe*, 410 U.S. at 165 (emphasis added); *Casey*, 505 U.S. at 879 (quoting *Roe*, same). *See also, e.g., Doe v. Bolton*, 410 U.S. 179, 192 (1973) ("[T]he medical judgment may be exercised in the light of all factors – physical, emotional, psychological, familial, and the woman's age – relevant to the well-being of the patient. All these factors

H.R. 3803 allows abortions only if necessary "to save the life of a pregnant woman," it would be unconstitutional *even if it applied only post-viability* (which it does not), because it does not allow abortions that may be necessary to preserve the health of the woman.

The Supreme Court's 2007 decision in *Gonzales v. Carhart*, 550 U.S. 124 (2007) ("*Carhart II*") did not change this. First, the Court in *Carhart II* expressly did not overrule any of its previous precedent. *See, e.g.*, *Carhart II*, 550 U.S. at 146 (stating that the Court was "apply[ing]" the standard set forth in *Casey*). While the federal statute at issue in *Carhart II* does not contain an exception to protect women's health, the Court was clear that it could nonetheless uphold that law because it reached just *one method* of abortion. Central to the Court's holding was the fact that other methods of abortion remained available in all instances. The Court explained that Congress could ban one method of abortion without a health exception "given the availability of other abortion procedures that are considered to be safe alternatives." 550 U.S. at 167; *see also id.* at 164 ("Alternatives are available to the prohibited procedure."); *id.* at 165 ("Here the Act allows, among other means, a commonly used and generally accepted method, so it does not construct a substantial obstacle to the abortion right.").

In contrast, H.R. 3803 bans *all* abortions after 20 weeks after fertilization – not just one method. Therefore, if a woman needs an abortion to protect her health, she would not have a safe alternative that would permit her to end her pregnancy, as Supreme Court precedent has required for nearly forty years.

II. THE ACT TRAMPLES ON THE RIGHTS OF D.C. RESIDENTS

H.R. 3803 is unprecedented: it would be the first time that the U.S. Congress has arrogated to itself the authority to prescribe rules of medical practice in one jurisdiction. The District of Columbia has a duly elected City Council and Mayor, and it is the province of those officials to consider and enact public policies governing D.C. residents. The sponsors of this bill ignore this fact and instead insist on using the District as a testing ground for their extreme and callous anti-choice agenda.

The 1973 Home Rule Act restored to the District of Columbia the basic democratic guarantee of consent of the governed, with a few limited exceptions. Dictating medical decisions and trampling the constitutional rights of D.C. women were not among those exceptions. While many

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may relate to health."); *Casey*, 505 U.S. at 882 ("It cannot be questioned that psychological well-being is a facet of health."); *Women's Med. Prof'l Corp. v. Voinovich*, 911 F. Supp. 1051, 1080-81 (S.D. Ohio 1995) (post-viability abortion restriction unconstitutional because "a state may not constitutionally limit the provision of abortions only to those situations in which a pregnant woman's *physical health* is threatened, because this impermissibly limits the physician's discretion to determine what measures are necessary to preserve her health") (emphasis added), *aff'd on other grounds*, 130 F.3d 187 (6th Cir. 1997).

anti-choice conservatives trumpet federalism and states' rights in other contexts, they are all too willing to set those principles aside and arrogantly impose cruel, dangerous and unconstitutional restrictions on women in the District of Columbia. The sponsors of this bill would be outraged if the United States Congress decided to reach into one of their districts – and no other – and impose rules restricting the delivery of medical care in that district. And yet that is exactly what they are doing to the District of Columbia.

III. THE ACT WOULD HARSHLY PENALIZE DOCTORS AND THREATEN THEM WITH LITIGATION

Under this bill, doctors could face two years in prison and fines of up to $250,000 for performing abortions after 20 weeks gestation, regardless of the circumstances, and could also be sued by their patients or their patients' families. These doctors are trying to provide the most medically appropriate care for their patients. As District resident Christy Zink explained in a statement about her medically necessary abortion: "I am horrified to think that the doctors who compassionately but objectively explained to us the prognosis and our options for medical treatment, and the doctor who helped us terminate the pregnancy, would be prosecuted as criminals under this law for providing basic medical care and expertise."[10]

The bill also allows a woman's husband, sibling, parent, and even any doctor who has ever treated the woman for any condition, to sue to prevent her from getting an abortion. It is stunning that Congress would even consider enacting legislation allowing someone else to sue a woman's doctor and to interfere with a decision to have a medical procedure that is a constitutionally protected right.

IV. THE ACT CRUELLY DISREGARDS THE AGONIZING CIRCUMSTANCES WOMEN FACE

H.R. 3803 would ban virtually all abortions after 20 weeks gestation, regardless of the woman's circumstances. For example, some women will learn at this point in pregnancy that the fetus she is carrying has severe abnormalities that make its survival highly unlikely or impossible; women and their families need time to consult with medical specialists and consider all their options in those difficult circumstances. This ban would impose an arbitrary and unnecessary deadline on those women, and would prevent physicians from providing the best medical care.

Consider some examples of how this law would harm women and their families:

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[10] "Statement of Christy Zink on Harmful Impact of HR 3803," February 21, 2012, available at http://prochoice.org/news/releases/20120221.html.

- Christy Zink, who lives and works in Washington, D.C., and her husband were devastated to learn, when Christy was 21 weeks pregnant, that their son was missing part of his brain. Had he survived childbirth, he would have experienced near-constant seizures and also near-constant pain. This bill would bar women in such tragic circumstances from taking the time they need to weigh all of the relevant information and ultimately make a difficult but compassionate decision to end the pregnancy.

- Tara Schleifer, a mother from Virginia, ended her pregnancy in the second trimester after learning the fetus had congenital heart and bowel defects that would cause debilitating pain and suffering. As reported, she "told the [Virginia] Education and Health Committee that any pain inflicted by her recent abortion undoubtedly paled in comparison to what her second child would have endured had he been brought into the world with myriad health issues: a heart defect requiring multiple open-heart surgeries, Down Syndrome and a bowel problem that would have required feeding through a tube. . . She ultimately concluded that having the baby would not only subject him to more suffering, but would leave the family financially and emotionally bankrupt and unfairly detract from the parenting of 3-year-old son Isaac."[11]

Every pregnancy is different. A one-size-fits-all ban callously ignores the very personal and often difficult circumstances surrounding a woman's decision to terminate a pregnancy. Women should be assisted by doctors, not politicians, in deciding the care they need, and they should not have those important decisions burdened by an arbitrary ticking clock.

V. THE ACT IS SCIENTIFICALLY UNSOUND

H.R. 3803's purported goal is to prevent fetal pain, but the "findings" in the bill deliberately obfuscate what the science actually shows. Britain's Royal College of Obstetricians and Gynecologists (RCOG) concluded in 2010 that nerve connections in the fetal brain are not sufficiently formed to allow pain perception before at least 24 weeks, and that pain is likely not experienced until birth.[12]

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[11] "Lawmakers Narrowly Reject Late-Term Abortion Ban In VA," CBS/DC, Feb. 12, 2012, available at: http://washington.cbslocal.com/2012/02/02/lawmakers-narrowly-reject-late-term-abortion-ban-in-va/.

[12] Available at http://www.rcog.org.uk/fetal-awareness-review-research-and-recommendations-practice.

Members of the medical faculty at the University of California San Francisco (UCSF), after reviewing the medical literature around fetal pain, also refute the key "findings" in H.R. 3803 and thus undercut the purported basis for this legislation.[13]

CONCLUSION

H.R. 3803 is merely the latest attempt by opponents of women's reproductive autonomy to put the nation on a path back to an era of illegal, unsafe abortion. With this bill, they target women of the District of Columbia, whose rights are regularly used as a political pawn, and seek to impose draconian and cruel restrictions on those women's most personal medical decisions. The bill fails to take into account women's individual circumstances, no matter how difficult, and makes no exceptions for threats to women's health. We urge the Subcommittee and the Congress to soundly reject H.R. 3803.

[13] Letter to Alabama State Legislature from Philip Darney, MD, MSc, and Mark Rosen, MD, University of California, San Francisco, March 30, 2011. The Alabama legislation and H.R. 3803 contain nearly identical legislative findings.

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Testimony of Willie Parker, MD, MPH, MSc
Board Member, Physicians for Reproductive Choice and Health
House Judiciary Committee
Subcommittee on the Constitution
May 17, 2012

Physicians for Reproductive Choice and Health (PRCH) is a doctor-led national advocacy organization that relies upon evidence-based medicine to promote sound reproductive health policies. PRCH opposes H.R. 3803, the "District of Columbia Pain-Capable Unborn Child Protection Act." This bill would ban abortion in the District of Columbia at 20 weeks after fertilization. This measure is clearly unconstitutional[i] and would harm women's health. Moreover, the bill is incredibly disrespectful of women, doctors, and the residents of the District of Columbia.

I am a board-certified obstetrician/gynecologist living and practicing medicine in the District. I have worked with Planned Parenthood of Metropolitan Washington, taught at the University of Hawaii John A. Burns School of Medicine, and served as an Epidemic Intelligence Service Officer with the Centers for Disease Control and Prevention. I received my medical degree from the University of Iowa, my master's degree in public health from the Harvard School of Public Health, and my master's degree in science in health services research from the University of Michigan, where I also completed a fellowship in family planning. I have more than 20 years of experience in women's health and have served on the PRCH board since 2007. I am pleased to submit this testimony in opposition to H.R. 3803 on behalf of PRCH.

I. H.R. 3803 Would Deny Women Needed Medical Care

Most abortions in the United States are provided early in pregnancy; roughly 12% of abortions occur at or after 13 weeks after a woman's last menstrual period (LMP). Only 1.4% of abortions occur at or after 21 weeks LMP.[ii] But some women will need abortion care later in pregnancy. H.R. 3803 would deny these women badly needed safe medical care.

While most women can look forward to a safe pregnancy, pregnancies can go terribly wrong. I remember caring for a senior staff member of a U.S. senator. At 23 1/2 weeks LMP, she discovered that her very desired pregnancy was complicated by a deadly fetal anomaly. She and her husband were distraught—this was their first child—but resolute that abortion was the right decision for them.[iii]

The difficult circumstances described above are not uncommon for abortions after 20 weeks post-fertilization, where discovery of complications and decision-making often occur. A physician in the PRCH network, Dr. Grace Shih in San Francisco, remembers one of her patients, whose water broke at 22 weeks LMP. Her pregnancy was doomed. Her wish was to have an abortion, as safely and quickly as possible, so that she could return home to her family and move forward.

Dr. Cat Cansino of Columbus, Ohio, cared for a patient whose pregnancy was diagnosed with a lethal fetal anomaly incompatible with life, after several consultations with high-risk obstetricians and neonatologists. Her patient shared with her how difficult it was to decide on abortion and also how hard it would have been to continue a pregnancy wondering when her baby would die while inside her.

Another physician, Dr. Aileen Gariepy of New Haven, Connecticut, took care of Angela, a 25-year-old woman with a very wanted pregnancy. She had come to Dr. Gariepy for a routine ultrasound at 23 weeks LMP. The ultrasound showed abnormalities, and later, the fetus was diagnosed with a lethal form of fetal skeletal dysplasia, a fatal bone disorder. Continuing the pregnancy would mean waiting for the fetus to die in utero, during labor, or immediately after delivery. Angela and her partner felt that the most compassionate thing to do was to end what they perceived as their baby's suffering and their own.

H.R. 3803 takes away decision-making from DC women and their doctors and replaces it with political judgment. Politicians should not insert their ideology into the most personal decisions of a woman and her family.

II. H.R. 3803 Lacks Adequate Exceptions, Contains Onerous Reporting Requirements, and Criminalizes Doctors' Care

H.R. 3803 only has a narrow exception for the life of a woman, inadequate exceptions to protect women's health, and no exceptions for rape, incest, or fetal anomalies. Many serious health conditions materialize or worsen later in pregnancy, such as placental bleeding. PRCH's consulting medical director, Dr. Anne Davis of New York, cared for a mother of two who was 22 weeks pregnant LMP. She had been bleeding throughout her pregnancy, but since this was a very desired pregnancy, she was waiting and hoping for the best. Her condition developed into placental abruption, which is where the placenta separates from the uterine wall, causing bleeding and depriving the fetus of oxygen. Her bleeding increased, and she was reaching the point where she would have suffered massive hemorrhage, shock, and death. Her pregnancy had to end. She survived and hopes to have more children.

I remember caring for a woman pregnant with her first child that developed a clotting disorder. The clotting disorder had destroyed her liver; she needed a liver transplant to save her life. She had to have an abortion so that she could have a liver transplant. H.R. 3803 would jeopardize the lives and health of all of these women. As discussed above, lethal fetal anomalies are also often not diagnosed until 20 weeks or later. H.R. 3803 would force women in the District to travel out of state (if they had the resources) or would deny them safe care altogether.

H.R. 3803 also contains an onerous and invasive reporting requirement. Any physician providing abortion care in the District would have to file reports on their patients. H.R. 3803 requires reporting of the gestational age of the pregnancy, the abortion method, and the age of the woman. The information would then be complied into a public report. While the legislation states that no information shall be included that could lead to the identification of patients, the language is inadequate and the legislation is silent as to the identification of doctors. When the CDC and many other states collect data, they require that statistics be provided in the aggregate. Aggregating statistics is necessary to protect the confidentiality of patients and physicians, and while the bill mentions confidentiality, it does not adequately ensure it.[iv] The District of Columbia is a small jurisdiction, which means that without sufficient protections, physicians could be singled out and identified, putting them at risk of violence and harassment. This is unacceptable.

This cruel legislation abandons and endangers women by criminalizing safe abortion. H.R. 3803 places my colleagues and me in the position of telling women that we cannot provide the medical care they need and deserve or risking civil and criminal penalties. Violation of this bill would result in fines and/or imprisonment of up to two years. The bill also grants the ability to sue for violations to relatives of the woman. These provisions are clearly intended to intimidate health care providers from providing abortion care.

III. Conclusion

Some states have already passed laws to ban abortions 20 weeks after fertilization. H.R. 3803 would create such a requirement in the District of Columbia. The DC government has not introduced this law—instead it has been introduced by Representative Trent Franks (R) of Arizona. Representative Franks and his cosponsors (none of whom represent the District of Columbia) purport to know what is best for the District, attempting to legislate an abortion restriction that the democratically elected local government has not supported or enacted.

There is a dearth of abortion providers in the United States. Eighty-seven percent of U.S. women live in a county where there is no access to abortion.[v] There is even less access for women who need abortion care after 20 weeks. My friend and colleague Dr. George Tiller of Kansas provided this needed care in Wichita until he was murdered in his Kansas church nearly three years ago. Dr. Tiller understood the needs of women in such complicated medical situations, making it his life's work to provide them with safe medical care.

The imposition of this ban in the District is meant not only to deprive DC women of safe and legal medical care, but also to intimidate and harass my colleagues and me who provide comprehensive and compassionate care to our patients. On behalf of PRCH, I urge you to vote against H.R. 3808.

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[i] The United States Supreme Court has long held that states may not ban abortion care before viability.

[ii] Centers for Disease Control and Prevention. Abortion Surveillance, United States, 2008. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6015a1.htm?s_cid=ss6015a1_w. Accessed May 16, 2012.

[iii] Compounding the horror of their situation were the delay and struggle they experienced when her federally funded health insurance initially refused to cover her abortion. I performed her procedure without complication, for which they were very grateful.

[iv] For example, the State of Alabama's statute specifies that the data be made available in the aggregate. (Alabama Statutes Section 22-9A-13.) The State of Michigan has a similar requirement: "the department shall make available annually *in aggregate* a statistical report summarizing the information submitted in each individual report required by this section [emphasis added]." (Michigan Public Health Code 333.2835).

[v] Guttmacher Institute, Facts on Induced Abortion in the United States, August 2011. Available at http://www.guttmacher.org/pubs/fb_induced_abortion.html. Accessed May 16, 2012.

District of Columbia Pain-Capable Unborn Child Protection Act (H.R.3803)
A Dangerous Restriction for Women

Testimony Presented by

Nancy Keenan
President

On Behalf of

NARAL Pro-Choice Arizona
NARAL Pro-Choice California
NARAL Pro-Choice Colorado
NARAL Pro-Choice Connecticut
NARAL Pro-Choice Maryland
NARAL Pro-Choice Massachusetts
NARAL Pro-Choice Minnesota
NARAL Pro-Choice Missouri
NARAL Pro-Choice Montana
NARAL Pro-Choice New Hampshire
NARAL Pro-Choice New Mexico
NARAL Pro-Choice New York
NARAL Pro-Choice North Carolina
NARAL Pro-Choice Ohio
NARAL Pro-Choice Oregon
NARAL Pro-Choice South Dakota
NARAL Pro-Choice Texas
NARAL Pro-Choice Virginia
NARAL Pro-Choice Washington
NARAL Pro-Choice Wisconsin
NARAL Pro-Choice Wyoming
Illinois Choice Action Team

U.S. House of Representatives
Committee on the Judiciary
Subcommittee on the Constitution

May 17, 2012

Members of the House Judiciary Subcommittee on the Constitution: I am honored to submit this testimony.

Today you are considering the District of Columbia Pain-Capable Unborn Child Protection Act (H.R.3803), introduced by Rep. Trent Franks (R-AZ). Under the auspices of concern about fetal pain, this misleadingly named bill singles out the District of Columbia for an unconstitutional ban on abortion care after 20 weeks, with no exceptions for a woman's health.

NARAL Pro-Choice America does not oppose post-viability bans that include appropriate exceptions for cases in which a woman's life and health are at risk. Yet 20-week abortion bans like H.R.3803 ignore the question of viability, and instead are meant as a direct challenge to the Supreme Court's ruling in *Roe v. Wade*. Additionally, by focusing this legislation solely on the District of Columbia, H.R.3803 is a clear affront to the principles of democracy and home rule. Ultimately this ban directly threatens the health of women in the most desperate of circumstances here in Washington, D.C. and attempts to set a precedent that would deny women their reproductive rights all across the country. NARAL Pro-Choice America opposes the Franks legislation and all other 20-week abortion bans.

The Franks Bill Is Part of a Coordinated Effort to Eliminate Access to Abortion Nationwide

While the Franks bill is the first attempt to pass a 20-week ban at the federal level, unfortunately its content is not unique. It is modeled on restrictions on abortion care that have been introduced in numerous states across the country. In 2010, Nebraska was the first state to pass a law instituting a ban on abortion after 20 weeks.[1] The following year, 15 states introduced 33 similar measures,[2] and by the end of 2011, five additional states—Alabama, Idaho, Indiana, Kansas, and Oklahoma—had enacted copycat bans.[3] So far in 2012, 20-week bans have become law in Arizona and Georgia, bringing the total number of states with such an extreme ban up to eight. None of these state laws has adequate exceptions to protect a woman's health or for cases of rape, incest, or fetal anomaly. The Franks bill is even more extreme than its state counterparts in that it contains no health exception at all.

Taken together, the Franks bill, the Nebraska law, and the subsequent bans in other states represent a new line of attack on women's reproductive rights, one which completely disregards the real-life circumstances of women and their families.

Some Women Need Later Abortion Care

The harsh reality is that while most women welcome pregnancy and can look forward to a safe childbirth, for some, pregnancy can be dangerous, and abortion restrictions without a health exception endanger these women. The Franks bill would force a woman to carry a pregnancy to term even if it would jeopardize her health, render her permanently disabled, or place her future fertility at risk.

H.R.3803 provides inadequate protections for the health of women. Take Vikki Stella for example, who, as a diabetic, discovered during her 32nd week of pregnancy that the fetus she was carrying suffered from several major anomalies and had no chance of survival. Because of Vikki's diabetes, her doctor determined that induced labor and Caesarian section were both riskier procedures for Vikki than an abortion. The procedure not only protected Vikki from immediate medical risks, but also ensured that she would be able to have children in the future.[4] The Franks bill would have precluded Vikki from choosing the best medical option for her situation.

Additionally, H.R.3803 includes no exception to protect women who are diagnosed with life-threatening conditions while pregnant. About one in 3,000 pregnant women has breast cancer during her pregnancy, and for these women, abortion care can be life-saving.[5] For instance, Jennifer Peterson was 35 and pregnant when she discovered a lump in her breast. Tests showed she had invasive breast cancer.[6] The cancer and its treatment, separate and apart from the pregnancy, were a threat to her health. A health exception for women like Jennifer recognizes the added health risk posed by pregnancy, both during the onset and treatment of her cancer. Yet, under the Franks bill, a woman like Jennifer would be forced to continue a pregnancy, even if she decided with her doctors that it would be dangerous to do so.

Abortion care can also be necessary for a woman whose pre-existing health condition could become fatal during pregnancy. Doctors report that many pregnant women with heart-valve disorders die each year from blood clots which, absent pregnancy, would not be life threatening.[7] A physician who specializes in maternal cardiac medicine said that there are "extreme pregnancy-associated risks" for women with these heart conditions. The doctor explained that: "A high risk of maternal mortality has implications not just for the mother but also for any potential baby and siblings at home. And even if she survives the pregnancy, the woman may have a reduced life expectancy or suffer from limited physical capacity."[8] For a woman presenting late in a pregnancy with a severe heart disorder, a health exception recognizes the totality of the risks she faces and allows her to make the best decision for her health, her life, and her family.

Exceptions must also account for the full range of health risks pregnant women face, including the tremendous emotional toll resulting from a pregnancy that encounters severe fetal anomaly. Women who seek abortion services after 20 weeks often have heartbreaking stories, such as that of Nebraska resident Danielle Deaver whose pregnancy suffered anhydramnios, a premature rupture of the membranes before the point of viability. Without sufficient amniotic fluid, the fetus likely would be born with a shortening of muscle tissue that results in the inability to move limbs, and the lungs were unlikely to develop beyond the 22-week point. In counsel with their doctor, Danielle and her husband explored every possible action to save the pregnancy. However, there was less than a 10 percent chance that, if born, the baby would be able to breathe on its own and only a two-percent chance the baby would be able to eat on its own.

This tragic situation was made even more difficult by the 2010 Nebraska law banning abortion after 20 weeks, which had gone into effect just two months prior. The new law meant that despite the extreme complications with her pregnancy, Danielle was unable to obtain an abortion. Eight days later, after Danielle endured intense pain and infection, their daughter was born and survived for just 15 minutes.[9] This heartbreaking story illustrates just one of many circumstances that a woman may face when a wanted pregnancy experiences complications. To also have to face a ban on abortion care after 20 weeks would cause more pain and anguish than any family should have to bear.

Finally, you will hear today from District of Columbia resident Christy Zink, who learned at 21 weeks that her pregnancy was suffering from severe fetal anomalies. The diagnosis she received meant that, if born, her baby would be in a state of near-constant seizures that would require numerous surgeries to remove what little of the brain matter remained. Christy and her husband made the difficult decision to terminate the pregnancy, a choice which would be illegal if the Franks bill were to become law.[10]

Women facing the direst of circumstances are among those who receive the 1.5 percent of abortions that occur after 20 weeks.[11] Sadly, with the murder of Dr. George Tiller in 2009 by an anti-choice extremist, women in these circumstances have fewer and fewer places to turn. H.R.3803 seeks to broaden the swath of women with no access to safe, legal abortion care after 20 weeks by banning it in the District of Columbia.

This Bill Lacks Other Important Exceptions

Further, the Franks bill and its state companions provide no exception for abortion in cases of rape or incest, even when the survivors of sexual violence are young girls. This is particularly harmful given that each year approximately 25,000 women in the United States become pregnant as a result of rape. Approximately 30 percent of rapes involve women under age 18.[12] Research by the Women's Reproductive Rights Assistance Project (WRRAP) found that girls 10-17 years of age accessed abortion care after 20 weeks — care that would be outlawed by H.R.3803 — more often than older women and that the women seeking WRRAP's assistance were more likely than the general population to report experiencing rape.[13] Some young survivors of sexual abuse or incest may access abortion care later in their pregnancies because they may not yet be as familiar with their bodies and may take some additional time to process the possibility of unintended pregnancy in addition to the trauma of rape. The youngest survivor documented in the WRRAP report was a 10-year-old victim of incest.[14] This ban does nothing to consider each woman's personal experiences and makes no exception for young women facing such trauma.

Twenty-Week Abortion Bans Are Blatantly Unconstitutional

The Supreme Court has long held that a woman has the unequivocal right to choose abortion care until the point of fetal viability. Under this standard, states may regulate abortion care, but

not ban it before viability.[15] However, 20-week abortion bans are not about post-viability abortion care. In fact, all eight states that have enacted 20-week bans already had post-viability bans in place at the time. Instead, by banning abortion at 20 weeks, the Franks legislation is clearly an unconstitutional pre-viability abortion ban. Its sponsors are attempting to use controversial and unsettled science to lure the court to discard the *Roe* framework entirely by moving away from the viability standard established in *Roe*.

In fact, state Sen. Mike Flood, the author of the Nebraska ban, openly acknowledges that his law "walks away from viability as a standard."[16] Anti-choice strategist Mary Spaulding Balch, attorney for the National Right to Life Committee, also has admitted that: "What I would like to bring to the attention of the court is, there is another line. This new knowledge is something the court has not looked at before and should look at."[17]

The shift in the Supreme Court's attitude towards a woman's right to choose as illustrated in its 2007 *Gonzalez v. Carhart* decision has opened the door to this round of attacks on women's reproductive health. By a slim 5-4 majority that included two conservative justices newly appointed by anti-choice President George W. Bush—Chief Justice John Roberts and Justice Samuel Alito—the court for the first time abandoned its holding that protections for a woman's health must always be paramount in any laws governing abortion. Now, anti-choice proponents of 20-week abortion bans, including Sen. Flood, readily admit that, "Absent the holding in *Gonzales*, I don't think Nebraska would have any ability to even propose a bill like this and see it held constitutional."[18]

Other opponents of reproductive choice have stated their goal even more plainly. In describing the Idaho 20-week ban passed in 2011, Lifenews.com reports that: "the law was put together in part to expand on the Supreme Court's decision upholding the partial-birth abortion ban Congress passed with the hopes of getting the same five Justices to agree to erode Roe v. Wade [sic] further."[19] The intent of the Franks bill is clear: to reverse a precedent that has protected women's reproductive rights for nearly four decades.

The Franks Bill Unfairly Targets the District of Columbia

Opponents of choice long have used the District of Columbia as an anti-choice proving ground. The Franks bill aimed at the reproductive rights of women living in the nation's capital is no different. In all but four of the last 23 years, anti-choice politicians have used the congressional appropriations process to impose a discriminatory restriction which bars the District of Columbia from using its own local funds to provide abortion care to its low-income residents, effectively narrowing the reproductive-health options of many poor women living in the District of Columbia. Similarly, the federal 20-week ban continues the anti-choice legacy of undermining home rule in the District of Columbia by supplanting the judgment of local leaders elected by District of Columbia citizens to serve their needs, and ultimately make a mockery of the democratic process in the nation's capital. Sponsors of H.R.3803 propose using

the health of women in the nation's capital as collateral in an attempt to undermine reproductive rights for women across the country.

Conclusion

The Franks ban on abortion care after 20 weeks is a blatant attempt to deny women their constitutional right to choose. H.R.3803 does not provide necessary exceptions to protect a woman's health and is a clear attack on the District of Columbia's home rule. The bill employs dubious rhetorical trappings to advance yet another restriction on a woman's right to choose, this one a dangerous limitation that could threaten the health of women in the most desperate of circumstances. As such, NARAL Pro-Choice America strongly opposes the Franks legislation and urges lawmakers to reject this harmful proposal.

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[1] L.B. 1103, 101st Legislature, 2nd session (Neb. 2010).

[2] These states are Alabama, Arkansas, Florida, Georgia, Iowa, Idaho, Indiana, Kansas, Kentucky, Minnesota, Mississippi, New Mexico, Oklahoma, Oregon, and South Carolina. NARAL Pro-Choice America Foundation, *Tracker Report* (internal document).

[3] NARAL Pro-Choice America Foundation, "Abortion Bans After 12 Weeks," *Who Decides? The Status of Women's Reproductive Rights in the United States at* http://www.prochoiceamerica.org/what-is-choice/maps-and-charts/map.jsp?mapID=16.; In addition, Minnesota's Gov. Mark Dayton (D) vetoed a measure that banned abortion at 20 weeks. (H.F. 936 87th Leg., 2011 1st Sess. (Minn. 2011).

[4] *Partial Birth Abortion Ban of 1995: Hearing on H.R.1833/S. 939 Before the Senate Comm. on the Judiciary*, 104th Cong. (1995) (testimony of Vikki Stella).

[5] The National Cancer Institute, U.S. National Institutes of Health, *General Information About Breast Cancer Treatment and Pregnancy* (Dec. 3, 2010), *at* http://www.cancer.gov/cancertopics/pdq/treatment/breast-cancer-and-pregnancy/HealthProfessional (last visited Oct. 27, 2011).

[6] Judy Foreman, *When Drugs Are The Only Choice For A Mother-To-Be*, Sept. 26, 2000, *at* http://judyforeman.com/columns/when-drugs-are-only-choice-mother-be (last visited Oct. 27, 2011).

[7] *Drug Fear Endangers Pregnant Women: Many Aren't Taking Medicine They Need*, USA TODAY, Dec. 12, 2000.

[8] Lisa Nainggolan, *Pregnant Pause: Evaluating Pregnant Women with Heart Disease* (Dec. 24, 2003), *at* http://www.theheart.org/article/124447.do (last visited Oct. 27, 2011).

[9] Jason Clayworth, *Her Baby Wasn't Expected to Live, But Nebraska Law Banned Abortion*, DES MOINES REGISTER, Mar. 6, 2011.

[10] Press Release, National Abortion Federation, *Statement of Christy Zink on Harmful Impact of HR 3803* (Feb. 21, 2012) *at* http://www.prochoice.org/news/releases/20120221.html (last visited Feb. 24, 2012).

[11] Guttmacher Institute, *In Brief: Facts on Induced Abortion in the United States*, Aug. 2011 *at* http://www.guttmacher.org/pubs/fb_induced_abortion.html (last visited Oct. 27, 2011).

[12] Felicia Stewart & James Trussell, *Prevention of Pregnancy Resulting from Rape: A Neglected Preventive Health Measure*, 19 AM. J. PREV. MED. 228, 228 (2000).

[13] NATIONAL NETWORK OF ABORTION FUNDS, *Abortion Funding: A Matter of Justice*, 2005, *at* http://fundabortionnow.org/sites/default/files/national_network_of_abortion_funds_-_abortion_funding_a_matter_of_justice.pdf.

[14] NATIONAL NETWORK OF ABORTION FUNDS, *Abortion Funding: A Matter of Justice*, 2005, *at* http://fundabortionnow.org/sites/default/files/national_network_of_abortion_funds_-_abortion_funding_a_matter_of_justice.pdf.

[15] *Roe*, 410 U.S. at 163-64. *Planned Parenthood of Southeastern Pennsylvania v. Casey*, 505 U.S. 833 (1992).

[16] Robert Barnes, *Tests of* Roe *More Frequent Since Justices Upheld Late-Term Abortion Ban in '07*, WASH.POST, Dec. 28, 2010 *at* http://www.washingtonpost.com/wp-dyn/content/article/2010/12/27/AR2010122703379.html (last visited Oct. 27, 2011).

[17] Bohon, Dave, *Nebraska Abortion Law Challenges* Roe v. Wade, NEW AMERICAN, April 17, 2010 *at* http://www.thenewamerican.com/index.php/culture/family/3340-nebraska-abortion-law-challenges-roe-v-wade (last visited Oct. 27, 2011).

[18] Robert Barnes, *Tests of* Roe *More Frequent Since Justices Upheld Late-Term Abortion Ban in '07*, WASH.POST, Dec. 28, 2010 *at* http://www.washingtonpost.com/wp-dyn/content/article/2010/12/27/AR2010122703379.html (last visited Oct. 27, 2011).

[19] Steven Ertelt, *Pro-Lifers Welcome First Fetal Pain Abortion Ban Lawsuit*, LifeNews.com, Sept. 1, 2011, http://www.lifenews.com/2011/09/01/pro-lifers-welcome-first-fetal-pain-abortion-ban-lawsuit/ (last visited May 15, 2012).

ACOG
THE AMERICAN CONGRESS
OF OBSTETRICIANS
AND GYNECOLOGISTS

June 18, 2012

ACOG STATEMENT ON HR 3803

The American Congress of Obstetricians and Gynecologists (ACOG) opposes HR 3803, the District of Columbia Pain-Capable Unborn Child Protection Act, and other legislative proposals that are not based on sound science or that attempt to prescribe how physicians should care for their patients. ACOG hopes our comments below will be helpful to the Committee in clarifying several inaccuracies in the bill's language and in testimony submitted in support of it. As the Nation's leading authority in women's health, our role is to ensure that policy proposals accurately reflect the best available medical knowledge.

Gestational Age
The bill's and supporters' language regarding post-fertilization age is vague and inaccurate. Obstetrician-gynecologists use last menstrual period (LMP) to date pregnancies. Post-fertilization dating is not an accurate substitute and should not be referenced in legislation.

Fetal Pain
The medical profession produced a rigorous scientific review of the available evidence on fetal pain in *Journal of the American Medical Association (JAMA)* in 2005[1]. The review concluded that fetal perception of pain is unlikely before the third trimester. No new studies since the publication of the *JAMA* paper have changed this dominant view of the medical profession. Supporters of HR 3803 only present studies which support the claim of fetal pain prior to the third trimester. When weighed together with other available information, including the *JAMA* study, the supporters' conclusion does not stand.

Fetal Viability
Most obstetrician-gynecologists understand fetal viability as occurring near 24 weeks gestation utilizing LMP dating. Submitted testimony by supporters of HR 3803 presents misleading evidence about fetal viability, especially in using post-fertilization age, instead of LMP dating, falsely implying high survival rates among neonates that are overwhelmingly pre-viable. While quoting survival of live-born infants in a June 2009 *JAMA* study, the testifier does not mention that the vast majority of infants born prior to 24 completed weeks (LMP) died prior to or during birth. In this study, 93% of infants at 22 weeks died, 66% at 23 weeks, and 40% at 24 weeks[2]. 91% of those that lived were admitted to the NICU.

Also not mentioned by the testifier is the fact that survival alone is not the only endpoint for neonatologists. Intact survival is. In this same study, 98% of infants born at 22 weeks (LMP) and 91% born at 23 weeks (LMP) had at least one major medical problem, such as hemorrhaging brain or bowel[2]. The American Academy of Pediatrics Committee on Fetus and Newborn states that "the incidence of moderate or severe neurodevelopmental disability in surviving children assessed at the age of 18 to 30 months is high (approximately 30 to 50%)[3]" and remains at that high level until 25 weeks (LMP). Babies delivered at these gestational ages often suffer hemorrhaging bowel, blindness, deafness, and stroke, as a result of their premature delivery.

Depression
Testimony submitted in support of HR 3803 asserts that women suffer from depression after abortion. A thorough review by the American Psychological Association in 2008 necessitates a more careful understanding of this issue:

> "[a]mong adult women who have an unplanned pregnancy, the relative risk of mental health problems is no greater if they have a single elective first-trimester abortion than if they deliver

that pregnancy. The evidence regarding the relative mental health risks associated with multiple abortions is more equivocal. Positive associations observed between multiple abortions and poorer mental health may be linked to co-occurring risks that predispose a woman to both multiple unwanted pregnancies and mental health problems. The few published studies that examined women's responses following an induced abortion due to fetal abnormality suggest that terminating a wanted pregnancy late in pregnancy due to fetal abnormality appears to be associated with negative psychological reactions equivalent to those experienced by women who miscarry a wanted pregnancy or who experience a stillbirth or death of a newborn, but less than those who deliver a child with life-threatening abnormalities."

ACOG opposes HR 3803 and strongly urges the Committee and the US Congress to closely examine and follow scientific facts and medical evidence in its consideration of this and other health care legislation. We stand ready to provide you with factual information on medical issues that come before the Committee, and hope you'll contact Nevena Minor, ACOG Director of Federal Affairs at nminor@acog.org, at any time.

References:

1. Lee SJ, Ralston HJP, Drey EA, Partridge JC, Rosen MA. Fetal pain: A systematic multidisciplinary review of the evidence. *JAMA* 2005; 294: 947-954.
2. EXPRESS group. One-year survival of extremely preterm infants after active perinatal care in Sweden. *JAMA* 2009; 301: 2225-2233.
3. MacDonald H & the Committee on Fetus and Newborn. Perinatal care at the threshold of viability. *Pediatrics* 2002; 110: 1024–1027.
4. APA Task Force on Mental Health and Abortion. (2008). *Report of the APA Task Force on Mental Health and Abortion.* Washington, D.C.: Author.
5. Dietz PM, Williams SB, Callaghan WM, Bachman DJ, Whitlock EP, Hornbrook MC. Clinically identified maternal depression before, during, and after pregnancies ending in live births. *Am J Psychiatry* 2007; 164: 1515–20.

Fetal Pain

A Systematic Multidisciplinary Review of the Evidence

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Context Proposed federal legislation would require physicians to inform women seeking abortions at 20 or more weeks after fertilization that the fetus feels pain and to offer anesthesia administered directly to the fetus. This article examines whether a fetus feels pain and if so, whether safe and effective techniques exist for providing direct fetal anesthesia or analgesia in the context of therapeutic procedures or abortion.

Evidence Acquisition Systematic search of PubMed for English-language articles focusing on human studies related to fetal pain, anesthesia, and analgesia. Included articles studied fetuses of less than 30 weeks' gestational age or specifically addressed fetal pain perception or nociception. Articles were reviewed for additional references. The search was performed without date limitations and was current as of June 6, 2005.

Evidence Synthesis Pain perception requires conscious recognition or awareness of a noxious stimulus. Neither withdrawal reflexes nor hormonal stress responses to invasive procedures prove the existence of fetal pain, because they can be elicited by nonpainful stimuli and occur without conscious cortical processing. Fetal awareness of noxious stimuli requires functional thalamocortical connections. Thalamocortical fibers begin appearing between 23 to 30 weeks' gestational age, while electroencephalography suggests the capacity for functional pain perception in preterm neonates probably does not exist before 29 or 30 weeks. For fetal surgery, women may receive general anesthesia and/or analgesics intended for placental transfer, and parenteral opioids may be administered to the fetus under direct or sonographic visualization. In these circumstances, administration of anesthesia and analgesia serves purposes unrelated to reduction of fetal pain, including inhibition of fetal movement, prevention of fetal hormonal stress responses, and induction of uterine atony.

Conclusions Evidence regarding the capacity for fetal pain is limited but indicates that fetal perception of pain is unlikely before the third trimester. Little or no evidence addresses the effectiveness of direct fetal anesthetic or analgesic techniques. Similarly, limited or no data exist on the safety of such techniques for pregnant women in the context of abortion. Anesthetic techniques currently used during fetal surgery are not directly applicable to abortion procedures.

JAMA. 2005;294:947-954 www.jama.com

Over the last several years, many states, including California, Kentucky, Minnesota, Montana, New York, Oregon, and Virginia, have considered legislation requiring physicians to inform women seeking abortions that the fetus feels pain and to offer fetal anesthesia. This year, Arkansas and Georgia enacted such statutes.[1,2] Currently, Congress is considering legislation requiring physicians to inform women seeking abortions 20 or more weeks after fertilization (ie, 22 weeks' gestational age) that the fetus has "physical structures necessary to experience pain," as evidenced by "draw[ing] away from surgical instruments." The physician must also offer anesthesia or analgesia "administered directly" to the fetus. Physicians who do not comply may be subject to substantial fines, license revocation, and civil suits for punitive damages.[3]

Although this legislation would not affect most US abortions because only 1.4% are performed at or after 21 weeks' gestational age,[4] this legislation raises important scientific, clinical, ethical, and policy issues. When does a fetus have the functional capacity to feel pain? If that capacity exists, what forms of anesthesia or analgesia are safe and effective for treating fetal pain? As a first step in answering these questions, we reviewed the literature on fetal pain and fetal anesthesia and analgesia.

CME available online at www.jama.com

EVIDENCE ACQUISITION

English-language articles involving human participants were searched using PubMed for (1) *fetal pain* (16 articles), *fetal anesthesia* (6 articles), and *fetal analgesia* (3 articles); (2) *fetus* and (*anesthesia or analgesia*) (1239 articles); (3) Medical Subject Headings (MeSH) *an-*

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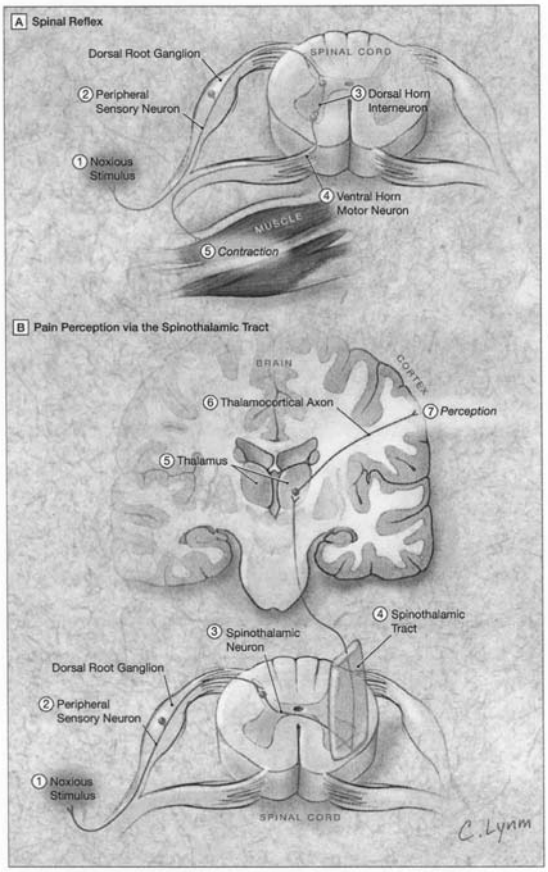
Figure. Spinal Reflex and Pain Perception Pathways



A, Reflex responses to noxious stimuli occur early in development, before thalamocortical circuits are functional; noxious stimuli trigger reflex movement without cortical involvement. Activated by a noxious stimulus (1), a peripheral sensory neuron (2) synapses on a dorsal horn interneuron (3) that in turn synapses on a ventral horn motor neuron (4), leading to reflex muscle contraction and limb withdrawal (5). B, Later in development, noxious stimuli (1) activate peripheral sensory neurons (2) that synapse on spinothalamic tract neurons (3), the axons of which extend up the spinal cord as the spinothalamic tract (4) to synapse on neurons of the thalamus (5). From here, thalamocortical axons synapse on cortical neurons, resulting in the conscious perception of pain.

algesics/administration and dosage and *fetus* (44 articles); (4) MeSH *anesthesia/administration and dosage* and *fetus* (0 articles); (5) (*neurodevelopment or development or anatomy*) and (*fetus or fetal*) and (*pain or nociception or noxious*) (306 articles); (6) (*thalamocortical or thalamus or cortex*) and (*fetus or fetal*) and (*pain or nociception or noxious*) (13 articles); (7) (*electroencephalog* or EEG or evoked potential*) and (*fetus or fetal or premature neonate or premature infant or preterm neonate or preterm infant*) and (*pain or nociception or noxious or conscious**) (7 articles); (8) *fetal* and *pain* and (*response or assessment or facial expression*) (112 articles); and (9) *facial expression* and (*fetus or fetal*) or ([*neonate or neonatal or infant*] and [*premature or preterm*]) and (*pain or nociception or noxious*) (360 articles). The search was performed without date limitations and was current as of June 6, 2005. From these search results, we excluded articles that did not study fetuses of less than 30 weeks' gestational age or that did not specifically address fetal pain perception or nociception. With a focus on topics addressed by earlier review articles on fetal pain, anesthesia, and analgesia, articles were reviewed for additional references.

EVIDENCE SYNTHESIS

What Is Pain?

Pain is a subjective sensory and emotional experience that requires the presence of consciousness to permit recognition of a stimulus as unpleasant.[5-7] Although pain is commonly associated with physical noxious stimuli, such as when one suffers a wound, pain is fundamentally a psychological construct that may exist even in the absence of physical stimuli, as seen in phantom limb pain.[5,7] The psychological nature of pain also distinguishes it from nociception, which involves physical activation of nociceptive pathways without the subjective emotional experience of pain.[5,8] For example, nociception without pain exists below the level of a spinal cord lesion, where reflex withdrawal from a noxious stimulus occurs without conscious perception of pain (FIGURE, A).[5]

Table. Anatomical and Functional Development of Nociception and Pain Perception Pathways

Anatomical/ Functional Characteristic	Description	Gestational Age, wk	Source
Peripheral cutaneous sensory receptors	Perioral cutaneous sensory receptors	7.5	Humphrey,[13] 1964
	Palmar cutaneous sensory receptors	10-10.5	
	Abdominal cutaneous sensory receptors	15	
Spinal cord	Spinal reflex arc in response to nonnoxious stimuli	8	Okado and Kojima,[14] 1984
	Neurons for nociception in dorsal root ganglion	19	Konstantinidou et al,[16] 1995
Thalamic afferents	Thalamic afferents reach subplate zone	20-22	Kostovic and Rakic,[18] 1990 Hevner,[17] 2000
	Thalamic afferents reach cortical plate	23-24	Kostovic and Rakic,[18] 1984 Kostovic and Goldman-Rakic,[19] 1983
Cortical function*	Somatosensory evoked potentials with distinct, constant components	29	Klimach and Cooke,[20] 1988 Hrbek et a,[21] 1973
	First electrocardiographic pattern denoting both wakefulness and active sleep	30	Cancy et al,[22] 2003 Torres and Anderson,[23] 1985

*Earliest evidence of functional thalamocortical connections required for conscious perception of pain.

Because pain is a psychological construct with emotional content, the experience of pain is modulated by changing emotional input and may need to be learned through life experience.[7,9,10] Regardless of whether the emotional content of pain is acquired, the psychological nature of pain presupposes the presence of functional thalamocortical circuitry required for conscious perception, as discussed below.

Fetal Capacity for Pain

Neuroanatomy and Development. Nociception may be characterized by reflex movement in response to a noxious stimulus, without cortical involvement or conscious pain perception. Nociception involves peripheral sensory receptors whose afferent fibers synapse in the spinal cord on interneurons, which synapse on motor neurons that also reside in the spinal cord. These motor neurons trigger muscle contraction, causing limb flexion away from a stimulus (Figure, A).[11]

In contrast, pain perception requires cortical recognition of the stimulus as unpleasant. Peripheral sensory receptor afferents synapse on spinal cord neurons, the axons of which project to the thalamus, which sends afferents to the cerebral cortex (Figure, B),[11] activating any number of cortical regions.[12] Sensory receptors and spinal cord synapses required for nociception develop earlier than the thalamocortical pathways required for conscious perception of pain (TABLE).

No human studies have directly examined the development of thalamocortical circuits associated with pain perception. The developmental age at which thalamic pain fibers reach the cortex has been inferred from studies of other thalamocortical circuits, which may or may not develop at the same time as thalamic fibers mediating cortical perception of pain.

These histological neurodevelopment studies typically describe fetal maturity in terms of developmental age, representing the number of weeks postovulation or postfertilization. Clinicians regularly use gestational age, representing weeks from the first day of the woman's last menstrual period. When referring to a fetus at the same point in development, the gestational age is approximately 2 weeks greater than the developmental age.

A histological study of the visual pathway in 8 human fetuses, each at a different developmental age, concluded that thalamic projections reach the visual cortex at 21 to 25 weeks' developmental age (approximately 23-27 weeks' gestational age), based on results from a fetus of 24 weeks' developmental age (26 weeks' gestational age).[18] A similar 7-fetus study found thalamic afferents reached the auditory cortical plate at 24 to 26 weeks' developmental age, with 1 specimen showing initial cortical plate penetration at 22 weeks' developmental age (24 weeks' gestational age).[24]

In a study of 8 human fetuses, mediodorsal thalamic afferents were first observed in the cortical plate at 22 weeks' developmental age (24 weeks' gestational age).[19] While connections between mediodorsal afferents and the anterior cingulate cortex[25] may be relevant to pain perception,[12,26] this study examined mediodorsal afferents to unspecified regions of the frontal cortex,[19] which serves numerous functions unrelated to pain perception.[19,27]

Another histological study of 12 specimens found that afferents from unspecified thalamic regions reached the developing prefrontal cortex in 1 preterm neonate of 27 weeks' developmental age, concluding that thalamic fibers begin entering the cortex between 26 and 28 weeks' developmental age (28 and 30 weeks' gestational age).[28] A different study found that thalamic afferents had not reached the somatosensory cortical plate by 22 weeks' developmental age (24 weeks' gestational age). By 24 weeks' developmental age (26 weeks' gestational age), the density of cortical plate synapses increased, although these were not necessarily from thalamic afferents.[10] Based on these studies, direct thalamocortical fibers that are not specific for pain begin to emerge between 21 and 28 weeks' developmental age (23 and 30 weeks' gestational age).

However, others have proposed that thalamocortical connections could also be established indirectly if thalamic afferents were to synapse on subplate neurons, which could synapse on cortical plate neurons.[29] The subplate is a transient fetal structure 1 layer deep to the cortical plate and serves as a "waiting compartment" for various afferents, including thalamic afferents, en route to the cortical plate.[16,29,30] The subplate recedes after 30 weeks' developmental age,[16,29] while the cortical plate matures into the 6 layers of the cerebral cortex.[28] In contrast to direct thalamocortical fibers, which are not visible until almost the third trimester, thalamic afferents begin to reach the somatosensory subplate at 18 weeks' developmental age (20 weeks' gestational age)[16] and the visual subplate at 20 to 22 weeks' gestational age.[17] These afferents appear morphologically mature enough to synapse with subplate neurons,[31] although no human study has shown that functional synapses exist between thalamic afferents and subplate neurons. Subplate neurons may synapse with cortical plate neurons and direct the growth of thalamic afferents to their final synaptic targets in the cortical plate.[29] Despite this developmental role, no human study has shown that synapses between subplate and cortical plate neurons convey information about pain perception from the thalamus to the developing cortex.

Electroencephalography. The histological presence of thalamocortical fibers is insufficient to establish capacity for pain perception. These anatomical structures must also be functional. Although no electroencephalographic "pain pattern" exists, electroencephalography may be one way of assessing general cortical function because electroencephalograms (EEGs) measure summated synaptic potentials from cortical neurons. However, EEG activity alone does not prove functionality, because neonates with anencephaly who lack functional neural tissue above the brainstem may still have EEG activity.[32]

Normal EEG patterns have been characterized for neonates as young as 24 weeks' postconceptional age (PCA) (ie, the gestational age plus number of weeks postpartum).[22] Electroencephalographic activity is normally asynchronous between the hemispheres and mostly discontinuous at less than 27 weeks' PCA,[23,33,34] becoming mostly continuous around 34 weeks' PCA.[23,34] Interhemispheric synchrony increases around 29 to 30 weeks' PCA, then declines, then increases again, reaching almost complete synchrony by term.[22,33] Given these baseline differences between neonatal and adult EEGs, patterns associated with impaired consciousness in adults[33,35] are inapplicable to the analysis of neonatal EEGs.

Some investigators contend that EEG patterns denoting wakefulness indicate when consciousness is first possible.[5,36] Wakefulness is a state of arousal mediated by the brainstem and thalamus in communication with the cortex.[5,22] In preterm neonates, the earliest EEG pattern representing wakefulness appears around 30 weeks' PCA.[22,23] However, wakefulness alone is insufficient to establish consciousness, as unconscious patients in a persistent vegetative state may also have wakeful EEGs.[5,36]

Somatosensory evoked potentials (SEPs) may also provide evidence of pain processing in the somatosensory cortex, although they are not used clinically to test pain pathways. SEPs test the dorsal column tract of the spinal cord, which transmits visceral pain sensation to the somatosensory cortex via the thalamus.[12] SEPs with distinct and constant N1 components of normal peak latency are present at 29 weeks' PCA, indicating that thalamic connections with the somatosensory cortex are functional at that time.[20,21]

Behavioral Studies. Although widely used to assess pain in neonates, withdrawal reflexes and facial movements do not necessarily represent conscious perception of pain. Full-term neonates exhibit a "cutaneous withdrawal reflex" that is activated at a threshold much lower than that which would produce discomfort in a child or adult.[37] This threshold increases with PCA, suggesting that the capacity of the neonate to distinguish between noxious and nonnoxious stimuli is maturing.[37] Furthermore, flexion withdrawal from tactile stimuli is a noncortical spinal reflex exhibited by infants with anencephaly[38] and by individuals in a persistent vegetative state[39] who lack cortical function.

Behavioral studies have also identified a distinct set of neonatal facial movements present during invasive procedures such as heel lancing but absent during noninvasive procedures.[40-46] These facial movements, which are similar to those of adults experiencing pain,[47,48] were evident in neonates at 28 to 30 weeks' PCA but not at 25 to 27 weeks' PCA.[10] Facial movements may not necessarily be cortically controlled.[19] One study found no difference in facial activity during heel lancing of neonates with and without significant cortical injury, suggesting that facial activity even around 32 weeks' PCA may not represent conscious perception of pain.[50]

Stress Responses. Hemodynamic and neuroendocrine changes in fetuses undergoing stressful procedures have also been used to infer pain perception.[51] As early as 16 weeks' gestational age, fetal cerebral blood flow increases during venipuncture and transfusions that access the fetal hepatic vein through the innervated fetal abdominal wall but not during venipuncture and transfusions involving the noninnervated umbilical cord.[52] Increased cerebral blood flow is not necessarily indicative of pain, as this response is thought to constitute a "brain sparing" mechanism associated with hypoxia[53] and intrauterine growth restriction.[54]

Other investigators measured increases in fetal plasma concentrations of cortisol, β-endorphin, and noradrenaline associated with intrauterine needling procedures, finding that increases during blood sampling from the hepatic vein were greater than those during sampling from the umbilical cord.[55,56] However, these neuroendocrine responses do not constitute evidence of fetal pain,

because the autonomic nervous system and hypothalamic-pituitary-adrenal axis mediate them without conscious cortical processing.[57] Additionally, these responses are not specific for painful stimuli. Plasma noradrenaline concentrations may increase after umbilical cord transfusion,[58] and plasma β-endorphin concentrations may increase after repeated cordocenteses.[59] Plasma cortisol and β-endorphin concentrations increase during innocuous activities such as exercise.[59] Moreover, in adults, neuroendocrine stress responses may persist despite well-controlled postoperative pain.[60]

Vital signs also have been used to assess neonatal pain.[42,43,45,51,61] However, heart rate, respiratory rate, and transcutaneous oxygen and carbon dioxide levels do not necessarily differ significantly between alcohol-swabbing and lancing the heels of preterm neonates.[40] Another group found that a similar proportion of neonates became hypoxic during tracheal suction, as well as during nonnoxious routine care such as washing and weighing.[62]

Fetal Anesthesia and Analgesia

Anesthetics and analgesics are commonly used to alleviate pain and discomfort. Despite ongoing debate regarding fetal capacity for pain, fetal anesthesia and analgesia are still warranted for surgical procedures undertaken to promote fetal health. When long-term fetal well-being is a central consideration, evidence of fetal pain is unnecessary to justify fetal anesthesia and analgesia because they serve other purposes unrelated to pain reduction, including (1) inhibiting fetal movement during a procedure[63-65]; (2) achieving uterine atony to improve surgical access to the fetus and to prevent contractions and placental separation[66-70]; (3) preventing hormonal stress responses associated with poor surgical outcomes in neonates[71,72]; and (4) preventing possible adverse effects on long-term neurodevelopment and behavioral responses to pain.[73-75]

These objectives are not applicable to abortions. Instead, beneficence toward the fetus represents the chief justification for using fetal anesthesia or analgesia during abortion—to relieve suffering if fetal pain exists. As with any clinical decision, thorough safety and risk-benefit analyses should be undertaken before performing an intervention. Because the principle of beneficence also requires the woman's physician to act in her best interests, potential fetal benefit must be weighed against real risks to the woman's health. The safety and effectiveness of proposed fetal anesthesia and analgesia techniques are discussed below.

General Anesthesia for Fetal Surgery. Fetal surgery involving laparotomy, hysterotomy, or both requires general or regional anesthesia.[66,76] Regional anesthesia, such as epidural anesthesia, does not anesthetize the fetus.[76] General anesthesia is more commonly used because it induces uterine atony and fetal immobilization.[65,77] Studies of inhalational agents in pregnant ewes determined that a dose capable of anesthetizing the ewe also anesthetized the fetus.[78] Administering fentanyl, pancuronium, or vecuronium to the fetus intramuscularly may supplement analgesia or immobilization.[64,65,77,79]

For pregnant women, general anesthesia is associated with increased morbidity and mortality, particularly because of airway-related complications[80-82] and increased risk of hemorrhage from uterine atony.[73] Historically, general anesthesia was used in abortions, even in the first trimester, until studies found that general anesthesia was a leading cause of abortion-related mortality.[83-85] In addition to safety concerns, general anesthesia increases the cost of abortion, making it prohibitively expensive for the majority of patients who pay out of pocket.[86]

Anesthesia and Analgesia in Minimally Invasive Fetal Procedures. In contrast to fetal surgery requiring regional or general anesthesia, minimally invasive fetal procedures do not involve maternal laparotomy or hysterotomy and instead use needles or endoscopy to access the fetus. For the sake of reducing pain, the increased risks of general anesthesia are unjustified for these procedures; adults typically undergo similar procedures with no analgesia or only local analgesia.[67] No established fetal analgesia protocol exists for these procedures, although 3 techniques have been proposed, namely, direct delivery of medications to the fetus, delivery of medications to the fetus via maternal intravenous infusion, and intra-amniotic delivery of medications.

Direct Delivery. One group has examined the effects of analgesics delivered directly to human fetuses during minimally invasive procedures.[87] Twenty-eight fetuses that received intravenous fentanyl before hepatic vein blood transfusions had diminished changes in plasma β-endorphin concentration and cerebral blood flow, compared with fetuses not receiving fentanyl. The cortisol response was not significantly decreased with fentanyl. The investigators did not examine risks for the woman, such as infection or uncontrolled bleeding.[76] Furthermore, reducing the stress response is distinct from reducing pain. For example, plasma glucose and cortisol concentrations may not differ significantly between adults with and without postoperative pain.[60]

Delivery via Maternal Intravenous Infusion. To achieve presumably effective fetal plasma concentrations of fentanyl by placental transfer, potentially unsafe doses would need to be administered to the woman.[88] Although standard doses of fentanyl are generally safe for maternal analgesia during labor,[89] fentanyl can pose serious risks such as hypoventilation if maternal doses are significantly increased to achieve more extensive placental transfer.[67,68] Severe maternal hypoventilation may require endotracheal intubation, which increases risks and costs for the woman, as described above.

No data exist on the dosing or efficacy of using medications such as diazepam and morphine for fetal analgesia via maternal intravenous infusion, although studies have characterized the placental transfer of these medications.[90-92] Two related studies found that

low-dose remifentanil via maternal intravenous infusion achieved fetal immobilization during laser coagulation of placental vessels.[93,94] However, immobilization is not the equivalent of pain reduction, and these procedures did not involve surgery on the fetus.

Intra-amniotic Delivery. Intra-amniotic injection would be technically simpler than direct fetal injection, although the drug must be absorbed through fetal membranes and skin. Intra-amniotic sufentanil injection in 10 pregnant ewes resulted in fetal plasma concentrations that would control postoperative pain in human adults.[95,96] Sufentanil concentrations in the ewes also reached adult human therapeutic concentrations without causing significant hemodynamic changes.[96] However, the study did not evaluate fetal response to noxious stimuli, and no data exist regarding safety or effectiveness in humans.

CONCLUSIONS

Pain is an emotional and psychological experience that requires conscious recognition of a noxious stimulus. Consequently, the capacity for conscious perception of pain can arise only after thalamocortical pathways begin to function, which may occur in the third trimester around 29 to 30 weeks' gestational age, based on the limited data available. Small-scale histological studies of human fetuses have found that thalamocortical fibers begin to form between 23 and 30 weeks' gestational age, but these studies did not specifically examine thalamocortical pathways active in pain perception.

While the presence of thalamocortical fibers is necessary for pain perception, their mere presence is insufficient—this pathway must also be functional. It has been proposed that transient, functional thalamocortical circuits may form via subplate neurons around midgestation, but no human study has demonstrated this early functionality. Instead, constant SEPs appear at 29 weeks' PCA, and EEG patterns denoting wakefulness appear around 30 weeks' PCA. Both of these tests of cortical function suggest that conscious perception of pain does not begin before the third trimester. Cutaneous withdrawal reflexes and hormonal stress responses present earlier in development are not explicit or sufficient evidence of pain perception because they are not specific to noxious stimuli and are not cortically mediated.

A variety of anesthetic and analgesic techniques have been used for fetal surgery, including maternal general anesthesia, regional anesthesia, and administration of medications for placental transfer to the fetus. However, these techniques are not necessarily applicable to abortions. Surgical procedures undertaken for fetal benefit use anesthesia to achieve objectives unrelated to pain control, such as uterine relaxation, fetal immobilization, and possible prevention of neuroendocrine stress responses associated with poor surgical outcomes. Thus, fetal anesthesia may be medically indicated for fetal surgery regardless of whether fetal pain exists.

In the context of abortion, fetal analgesia would be used solely for beneficence toward the fetus, assuming fetal pain exists. This interest must be considered in concert with maternal safety and fetal effectiveness of any proposed anesthetic or analgesic technique. For instance, general anesthesia increases abortion morbidity and mortality for women and substantially increases the cost of abortion. Although placental transfer of many opioids and sedative-hypnotics has been determined, the maternal dose required for fetal analgesia is unknown, as is the safety for women at such doses. Furthermore, no established protocols exist for administering anesthesia or analgesia directly to the fetus for minimally invasive fetal procedures or abortions. Experimental techniques, such as administration of fentanyl directly to the fetus and intra-amniotic injection of sufentanil in pregnant ewes, have not been shown to decrease fetal pain and are of unknown safety in humans.

Because pain perception probably does not function before the third trimester, discussions of fetal pain for abortions performed before the end of the second trimester should be noncompulsory. Fetal anesthesia or analgesia should not be recommended or routinely offered for abortion because current experimental techniques provide unknown fetal benefit and may increase risks for the woman. Instead, further research should focus on when pain-related thalamocortical pathways become functional in humans. If the fetus can feel pain, additional research may lead to effective fetal anesthesia or analgesia techniques that are also safe for women.

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REFERENCES

1. Unborn Child Pain Awareness and Prevention Act of 2005. To be codified at Ark Code Ann §§20-16-1101 to 1111.
2. Woman's Right to Know Act. To be codified at Ga Code Ann §31-9A-4.
3. Unborn Child Pain Awareness Act, S51, 109th Cong (2005).
4. Strauss LT, Herndon J, Chang J, et al. Abortion surveillance—United States, 2001. *MMWR Surveill Summ*. 2004;53:1-32.
5. Benatar D, Benatar M. A pain in the fetus: toward ending confusion about fetal pain. *Bioethics*. 2001;15:57-76.
6. Glover V, Fisk NM. Fetal pain: implications for research and practice. *Br J Obstet Gynaecol*. 1999;106:881-886.
7. International Association for the Study of Pain. IASP Pain Terminology. 2004. Available at: http://www.iasp-pain.org/terms-p.html. Accessed May 2, 2005.
8. Anand KJ, Hickey PR. Pain and its effects in the human neonate and fetus. *N Engl J Med*. 1987;317:1321-1329.
9. Derbyshire SW. Locating the beginnings of pain. *Bioethics*. 1999;13:1-31.
10. Derbyshire SW. Fetal pain: an infantile debate. *Bioethics*. 2001;15:77-84.
11. Fitzgerald M, Howard RF. The neurobiologic basis of pediatric pain. In: Schechter NL, Berde CB, Yaster M, eds. *Pain in Infants, Children, and Adolescents*. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2003:19-42.
12. Strigo IA, Duncan GH, Boivin M, Bushnell MC. Differentiation of visceral and cutaneous pain in the human brain. *J Neurophysiol*. 2003;89:3294-3303.
13. Humphrey T. Some correlations between the appearance of fetal reflexes and the development of the nervous system. *Prog Brain Res*. 1964;4:93-135.
14. Okado N, Kojima T. Ontogeny of the central nervous system: neurogenesis, fibre connection, synaptogenesis and myelination in the spinal cord. In: Prechtl HFR, ed. *Clinics in Developmental Medicine: Continuity of Neural Functions From Prenatal to Postnatal Life*. Vol 94. Philadelphia, Pa: JB Lippincott Co; 1984:31-45.
15. Konstantinidou AD, Silos-Santiago I, Flaris N, Snider WD. Development of the primary afferent projection

in human spinal cord. *J Comp Neurol*. 1995;354:11-12.
16. Kostovic I, Rakic P. Developmental history of the transient subplate zone in the visual and somatosensory cortex of the macaque monkey and human brain. *J Comp Neurol*. 1990;297:441-470.
17. Hevner RF. Development of connections in the human visual system during fetal mid-gestation: a DiI-tracing study. *J Neuropathol Exp Neurol*. 2000;59:385-392.
18. Kostovic I, Rakic P. Development of prestriate visual projections in the monkey and human fetal cerebrum revealed by transient cholinesterase staining. *J Neurosci*. 1984;4:25-42.
19. Kostovic I, Goldman-Rakic PS. Transient cholinesterase staining in the mediodorsal nucleus of the thalamus and its connections in the developing human and monkey brain. *J Comp Neurol*. 1983;219:431-447.
20. Klimach VJ, Cooke RW. Maturation of the neonatal somatosensory evoked response in preterm infants. *Dev Med Child Neurol*. 1988;30:208-214.
21. Hrbek A, Karlberg P, Olsson T. Development of visual and somatosensory evoked responses in preterm newborn infants. *Electroencephalogr Clin Neurophysiol*. 1973;34:225-232.
22. Clancy RR, Bergqvist AGC, Dlugos DJ. Neonatal electroencephalography. In: Ebersole JS, Pedley TA, eds. *Current Practice of Clinical Electroencephalography*. 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2003:160-234.
23. Torres F, Anderson C. The normal EEG of the human newborn. *J Clin Neurophysiol*. 1985;2:89-103.
24. Krmpotic-Nemanic J, Kostovic I, Kelovic Z, Nemanic D, Mrzljak L. Development of the human fetal auditory cortex: growth of afferent fibres. *Acta Anat (Basel)*. 1983;116:69-73.
25. Vogt BA, Rosene DL, Pandya DN. Thalamic and cortical afferents differentiate anterior from posterior cingulate cortex in the monkey. *Science*. 1979;204:205-207.
26. Schnitzler A, Ploner M. Neurophysiology and functional neuroanatomy of pain perception. *J Clin Neurophysiol*. 2000;17:592-603.
27. Barbas H. Connections underlying the synthesis of cognition, memory, and emotion in primate prefrontal cortices. *Brain Res Bull*. 2000;52:319-330.
28. Mrzljak L, Uylings HB, Kostovic I, Van Eden CG. Prenatal development of neurons in the human prefrontal cortex. I: a qualitative Golgi study. *J Comp Neurol*. 1988;271:355-386.
29. Kostovic I, Judas M. Correlation between the sequential ingrowth of afferents and transient patterns of cortical lamination in preterm infants. *Anat Rec*. 2002;267:1-6.
30. Ulfig N, Neudorfer F, Bohl J. Transient structures of the human fetal brain: subplate, thalamic reticular complex, ganglionic eminence. *Histol Histopathol*. 2000;15:771-790.
31. Kostovic I, Judas M, Petanjek Z, Simic G. Ontogenesis of goal-directed behavior: anatomo-functional considerations. *Int J Psychophysiol*. 1995;19:85-102.
32. Schenk VW, De Vlieger M, Hamersma K, De Weerdt J. Two rhombencephalic anencephalics: a clinico-pathological and electroencephalographic study. *Brain*. 1968;91:497-506.
33. Fisch BJ, Spehlmann R. *Fisch and Spehlmann's EEG Primer: Basic Principles of Digital and Analog EEG*. 3rd ed. New York, NY: Elsevier; 1999.
34. Scher MS. Electroencephalography of the newborn: normal and abnormal features. In: Niedermeyer E, Lopes da Silva FH, eds. *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. 4th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999:896-946.
35. Sharbrough FW. Nonspecific abnormal EEG patterns. In: Niedermeyer E, Lopes da Silva FH, eds. *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. 4th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999:215-234.
36. Burgess JA, Tawia SA. When did you first begin to feel it?—locating the beginning of human consciousness. *Bioethics*. 1996;10:1-26.
37. Andrews K, Fitzgerald M. The cutaneous withdrawal reflex in human neonates: sensitization, receptive fields, and the effects of contralateral stimulation. *Pain*. 1994;56:95-101.
38. Ashwal S, Peabody JL, Schneider S, Tomasi LG, Emery JR, Peckham N. Anencephaly: clinical determination of brain death and neuropathologic studies. *Pediatr Neurol*. 1990;6:233-239.
39. Pilon M, Sullivan SJ. Motor profile of patients in minimally responsive and persistent vegetative states. *Brain Inj*. 1996;10:421-437.
40. Craig KD, Whitfield MF, Grunau RV, Linton J, Hadjistavropoulos HD. Pain in the preterm neonate: behavioural and physiological indices. *Pain*. 1993;52:287-299.
41. Johnston CC, Stevens BJ, Yang F, Horton L. Differential response to pain by very premature neonates. *Pain*. 1995;61:471-479.
42. Johnston CC, Stevens B, Yang F, Horton L. Developmental changes in response to heelstick in preterm infants: a prospective cohort study. *Dev Med Child Neurol*. 1996;38:438-445.
43. Lindh V, Wiklund U, Sandman PO, Hakansson S. Assessment of acute pain in preterm infants by evaluation of facial expression and frequency domain analysis of heart rate variability. *Early Hum Dev*. 1997;48:131-142.
44. Hadjistavropoulos HD, Craig KD, Grunau RE, Whitfield MF. Judging pain in infants: behavioural, contextual, and developmental determinants. *Pain*. 1997;73:319-324.
45. Goubet N, Clifton RK, Shah B. Learning about pain in preterm newborns. *J Dev Behav Pediatr*. 2001;22:418-424.
46. Xie C, Yang L, Zhao P, Zhang X. Response to pain by different gestational age neonates. *J Huazhong Univ Sci Technolog Med Sci*. 2002;22:84-86.
47. Craig KD, Prkachin KM, Grunau RE. Facial expression of pain. In: Turk DC, Melzack R, eds. *Handbook of Pain Assessment*. 2nd ed. New York, NY: Guilford Press; 2001:153-169.
48. Craig KD, Hadjistavropoulos HD, Grunau RV, Whitfield MF. A comparison of two measures of facial activity during pain in the newborn child. *J Pediatr Psychol*. 1994;19:305-318.
49. Waxman SG. Clinical observations on the emotional motor system. *Prog Brain Res*. 1996;107:595-604.
50. Oberlander TF, Grunau RE, Fitzgerald C, Whitfield MF. Does parenchymal brain injury affect biobehavioral pain responses in very low birth weight infants at 32 weeks' postconceptional age? *Pediatrics*. 2002;110:570-576.
51. Franck LS, Miaskowski C. Measurement of neonatal responses to painful stimuli: a research review. *J Pain Symptom Manage*. 1997;14:343-378.
52. Teixeira JM, Glover V, Fisk NM. Acute cerebral redistribubon in response to invasive procedures in the human fetus. *Am J Obstet Gynecol*. 1999;181:1018-1025.
53. Woo JS, Liang ST, Lo RL, Chan FY. Middle cerebral artery Doppler flow velocity waveforms. *Obstet Gynecol*. 1987;70:613-616.
54. Wladimiroff JW, vd Wijngaard JA, Degani S, Noordam MJ, van Eyck J, Tonge HM. Cerebral and umbilical arterial blood flow velocity waveforms in normal and growth-retarded pregnancies. *Obstet Gynecol*. 1987;69:705-709.
55. Giannakoulopoulos X, Sepulveda W, Kourtis P, Glover V, Fisk NM. Fetal plasma cortisol and beta-endorphin response to intrauterine needling. *Lancet*. 1994;344:77-81.
56. Giannakoulopoulos X, Teixeira J, Fisk N, Glover V. Human fetal and maternal noradrenaline responses to invasive procedures. *Pediatr Res*. 1999;45:494-499.
57. Carrasco GA, Van de Kar LD. Neuroendocrine pharmacology of stress. *Eur J Pharmacol*. 2003;463:235-272.
58. Radunovic N, Lockwood CJ, Ghidini A, Alvarez M, Berkowitz RL. Is fetal blood sampling associated with increased beta-endorphin release into the fetal circulation? *Am J Perinatol*. 1993;10:112-114.
59. Williams RH, Larsen PR. *Williams Textbook of Endocrinology*. 10th ed. Philadelphia, Pa: Saunders; 2003.
60. Schulze S, Roikjaer O, Hasselstrom L, Jensen NH, Kehlet H. Epidural bupivacaine and morphine plus systemic indomethacin eliminates pain but not systemic response and convalescence after cholecystectomy. *Surgery*. 1988;103:321-327.
61. Porter FL, Wolf CM, Miller JP. Procedural pain in newborn infants: the influence of intensity and development. *Pediatrics*. 1999;104:e13.
62. Pokela ML. Pain relief can reduce hypoxemia in distressed neonates during routine treatment procedures. *Pediatrics*. 1994;93:379-383.
63. Seeds JW, Corke BC, Spielman FJ. Prevention of fetal movement during invasive procedures with pancuronium bromide. *Am J Obstet Gynecol*. 1986;155:818-819.
64. Rosen MA. Anesthesia for fetal procedures and surgery. *Yonsei Med J*. 2001;42:669-680.
65. Cauldwell CB. Anesthesia for fetal surgery. *Anesthesiol Clin North America*. 2002;20:211-226.
66. Rosen MA. Anesthesia for procedures involving the fetus. *Semin Perinatol*. 1991;15:410-417.
67. Smith RP, Gitau R, Glover V, Fisk NM. Pain and stress in the human fetus. *Eur J Obstet Gynecol Reprod Biol*. 2000;92:161-165.
68. White MC, Wolf AR. Pain and stress in the human fetus. *Best Pract Res Clin Anaesthesiol*. 2004;18:205-220.
69. Rosen MA. Anesthesia and tocolysis for fetal intervention. In: Harrison MR, Golbus MS, Filly RA, eds. *The Unborn Patient: Prenatal Diagnosis and Treatment*. Orlando, Fla: Grune & Stratton; 1984:417-433.
70. Schwarz U, Galinkin JL. Anesthesia for fetal surgery. *Semin Pediatr Surg*. 2003;12:196-201.
71. Anand KJ, Hickey PR. Halothane-morphine compared with high-dose sufentanil for anesthesia and postoperative analgesia in neonatal cardiac surgery. *N Engl J Med*. 1992;326:1-9.
72. Anand KJ, Sippell WG, Aynsley-Green A. Randomised trial of fentanyl anaesthesia in preterm babies undergoing surgery: effects on the stress response. *Lancet*. 1987;1:62-66.
73. Johnston CC, Stevens BJ. Experience in a neonatal intensive care unit affects pain response. *Pediatrics*. 1996;98:925-930.
74. Taddio A, Katz J, Ilersich AL, Koren G. Effect of neonatal circumcision on pain response during subsequent routine vaccination. *Lancet*. 1997;349:599-603.
75. Taylor A, Fisk NM, Glover V. Mode of delivery and subsequent stress response. *Lancet*. 2000;355:120.
76. Myers LB, Buisch LA, Hess P, Miller NM. Fetal endoscopic surgery: indications and anaesthetic management. *Best Pract Res Clin Anaesthesiol*. 2004;18:231-258.
77. Myers LB, Cohen D, Galinkin J, Gaiser R, Kurth CD. Anaesthesia for fetal surgery. *Paedratr Anaesth*. 2002;12:569-578.
78. Gregory GA, Wade JG, Beihl DR, Ong BY, Sitar DS. Fetal anesthetic requirement (MAC) for halothane. *Anesth Analg*. 1983;62:9-14.
79. Gaiser RR, Kurth CD. Anesthetic considerations for fetal surgery. *Semin Perinatol*. 1999;23:507-514.

80. Clyburn PA. Early thoughts on "Why Mothers Die 2000-2002." *Anaesthesia*. 2004;59:1157-1159.
81. Munnur U, Suresh MS. Airway problems in pregnancy. *Crit Care Clin*. 2004;20:617-642.
82. Royal College of Obstetricians and Gynaecologists. *Confidential Enquiry into Maternal and Child Health: Why Mothers Die 2000-2002: Report on Confidential Enquiries into Maternal Deaths in the United Kingdom*. London, England: RCOG Press; 2004.
83. Atrash HK, Cheek TG, Hogue CJ. Legal abortion mortality and general anesthesia. *Am J Obstet Gynecol*. 1988;158:420-424.
84. Atrash HK, MacKay HT, Binkin NJ, Hogue CJ. Legal abortion mortality in the United States: 1972 to 1982. *Am J Obstet Gynecol*. 1987;156:605-612.
85. Bartlett LA, Berg CJ, Shulman HB, et al. Risk factors for legal induced abortion-related mortality in the United States. *Obstet Gynecol*. 2004;103:729-737.
86. Henshaw SK, Finer LB. The accessibility of abortion services in the United States, 2001. *Perspect Sex Reprod Health*. 2003;35:16-24.
87. Fisk NM, Citau R, Teixeira JM, Giannakoulopoulos X, Cameron AD, Glover VA. Effect of direct fetal opioid analgesia on fetal hormonal and hemodynamic stress response to intrauterine needling. *Anesthesiology*. 2001;95:828-835.
88. Loftus JR, Hill H, Cohen SE. Placental transfer and neonatal effects of epidural sufentanil and fentanyl administered with bupivacaine during labor. *Anesthesiology*. 1995;83:308-308.
89. Rayburn W, Rathke A, Leuschen MP, Chleborad J, Weidner W. Fentanyl citrate analgesia during labor. *Am J Obstet Gynecol*. 1989;161:202-206.
90. Jauniaux E, Jurkovic D, Lees C, Campbell S, Gulbis B. In-vivo study of diazepam transfer across the first trimester human placenta. *Hum Reprod*. 1996;11:889-892.
91. Haram K, Bakke OM. Diazepam as an induction agent for caesarean section: a clinical and pharmacokinetic study of fetal drug exposure. *Br J Obstet Gynaecol*. 1980;87:506-512.
92. Gerdin E, Rane A, Lindberg B. Transplacental transfer of morphine in man. *J Perinat Med*. 1990;18:305-312.
93. Missant C, Van Schoubroeck D, Deprest J, Devlieger R, Teunkens A, Van de Velde M. Remifentanil for foetal immobilisation and maternal sedation during endoscopic treatment of twin-to-twin transfusion syndrome: a preliminary dose-finding study. *Acta Anaesthesiol Belg*. 2004;55:239-244.
94. Van de Velde M, Van Schoubroeck D, Lewi LE, et al. Remifentanil for fetal immobilization and maternal sedation during fetoscopic surgery: a randomized, double blind comparison with diazepam. *Anesth Analg*. 2005;101:251-258.
95. Lehmann KA, Gerhard A, Horrichs-Haermeyer G, Grond S, Zech D. Postoperative patient-controlled analgesia with sufentanil: analgesic efficacy and minimum effective concentrations. *Acta Anaesthesiol Scand*. 1991;35:221-226.
96. Strumper D, Durieux ME, Gogarten W, Van Aken H, Hartleb K, Marcus MA. Fetal plasma concentrations after intraamniotic sufentanil in chronically instrumented pregnant sheep. *Anesthesiology*. 2003;98:1400-1406.

> The polymorphic visions of the eyes and the spirit are contained in the uniform lines of small or capital letters, periods, commas, parentheses—pages of signs, packed as closely together as grains of sand, representing the many-colored spectacle of the world on a surface that is always the same and always different, like dunes shifted by the desert wind.
> —Italo Calvino (1923-1985)

Material submitted by the Honorable Robert C. "Bobby" Scott, a Representative in Congress from the State of Virginia, and Member, Subcommittee on the Constitution

A Perspective on Later Abortion... From Someone Who Does Them

By Willie Parker, MD

I AM INTRIGUED BY SOME REPRODUCTIVE rights advocates' increasing willingness to search for "common ground" with abortion opponents, evidenced by a recent conference convened with this purpose at a major university. Prior to the conference, one of its organizers, long-time reproductive rights supporter and former Catholics for Choice president Frances Kissling, expressed sentiments representative of this disturbingly conciliatory tone:

> "As long as women have an adequate amount of time to make a decision, and there are provisions for unusual circumstances that occur after that time, I would be satisfied [with early gestational age limits to abortion].... Women have an obligation to make this decision as soon as they possibly can."

In short, the abortion debate has come to include abortion supporters and opponents bargaining about restricting second-trimester abortion as a means of seeking common ground. While I applaud efforts towards a more civil public discourse in principle, as a provider of second-trimester abortion services, I find this trend problematic and dangerous to the health interests of women. I am also troubled by the question—to whom, other than themselves, are women obligated "to make their decision as soon as they possibly can"?

Apparently recognizing that termination of pregnancy won't be outlawed any time soon, abortion opponents are willing to engage in dialogues that—while appearing to progress towards a more civil exchange with abortion supporters—unwittingly enlist the energies of abortion rights activists for the restriction of those rights. These conversations subtly endorse the parsing away of this fundamental human right, ironically beginning with women in their second trimester, who often have the most compelling need to have an abortion in the first place. As is common in discussions

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of abortion, absent from these dialogues are the voices of the women and families that are affected—the very women who are and will be denied access to what is oftentimes a health-related decision.

The lives of these women and their families are what compelled me to add abortion care to my practice, mid-career, when I was no longer able to weigh the life of a pre-viable or lethally-flawed, viable fetus equally with the life of the woman sitting before me. My intent here is to share why I provide abortions. The times in which we live call for a thoughtful, compassionate, evidence-based approach to women's healthcare that should empower healthcare providers to include abortion in their practice—second-trimester abortions included—because of the women who, in the absence of these services, would die unnecessarily.

I did not provide abortions for the first 12 years of my career as an obstetrician/gynecologist, even though my work allowed me to see first-hand the reproductive dilemmas and outcomes that women and families face. While recognizing that abortion was a need in my patients' lives, I grappled with the morality of providing them, as I came from a traditional religious background that considered abortion to be wrong. It is said that when you grapple with your conscience and lose—you actually win. I "lost" that 12-year battle about whether or not to provide abortions while listening to a sermon by Dr. Martin Luther King, Jr.

Dr. King related the story of the Good Samaritan to encourage compassionate action on behalf of others. The story tells of an injured traveler who was ignored by passersby until one person, the Samaritan, stopped to help. According to Dr. King, what made the Good Samaritan "good" was his refusal to place himself first, asking instead, "What will happen to this person if I don't stop to help him?" Similarly, I asked the simple question of myself, "What happens to women who seek abortion if I don't serve them?" This radicalized me, leaving me more concerned about the unnecessary peril to women when safe abortion services are not available than about what would happen to me if I helped women in this way. It was at that point—some eight years ago—that I began to perform abortions, compelled by women's situations and moved to action by their need, and by my respect for their moral agency to make such a decision.

The women I see in these situations are pregnant and they can't be or don't want to be. They are resolving dilemmas created by circumstances unique to their private lives, and certainly unknown to their critics who judge from afar.

The stories of the women who come to me are what move me to overlook the well-established danger of antiabortion violence to do this work. Approximately one in three women in the US will terminate a pregnancy in her lifetime. While the epidemiology of women who have abortions gives a general impression of who they are—40 percent of US pregnancies are unplanned, with about half of this number unwanted—it is the specific realities of women who seek abortion, especially in the second trimester, that best inform us. The stories of the following women and girls that I have cared for provide a small glimpse into their reality of unplanned, unwanted or wanted but lethally-flawed pregnancies:

An 11-year-old was discovered by her grandmother to be 19 weeks pregnant the day before she was to start sixth grade. A trip to an emergency room confirmed the pregnancy, leading the family to seek abortion services. While the young lady refused to name who impregnated her, our best judgment was that it did not indicate incest. In talking to her to determine "who" desired the termination, she did not want to be pregnant and was not being coerced, but the stark reality of just how young she was became explicit when she expressed her chief concern: she had missed three days of school and wanted to be with her friends. I safely terminated her pregnancy and restored her childhood by allowing her to have the only concerns an 11-year-old should have.

A 13-year-old girl was a victim of incest by her uncle who had lived with the family for six months. By the time the girl's mother discovered her pregnancy, she was 17 weeks along. Her quiet demeanor, interpreted by her mother as ideal behavior, unfortunately delayed the detection of her pregnancy. We performed her abortion, but the family was understandably deeply shocked by the circumstances of the abortion.

A 32-year-old attorney, senior staff for a prominent US senator, came in with a desired pregnancy at 20 weeks, complicated by a lethal fetal anomaly. By the time diagnosis was confirmed, she was 23 ½ weeks. She and her husband were distraught, as this was their first child, but resolute that this was the right decision for them. Compounding the horror of their situation were the delay and struggle they experienced when her federally-funded health insurance initially refused to cover her abortion. I performed her procedure without complication, for which they were effusively grateful.

The difficult circumstances described above are typical for second-trimester

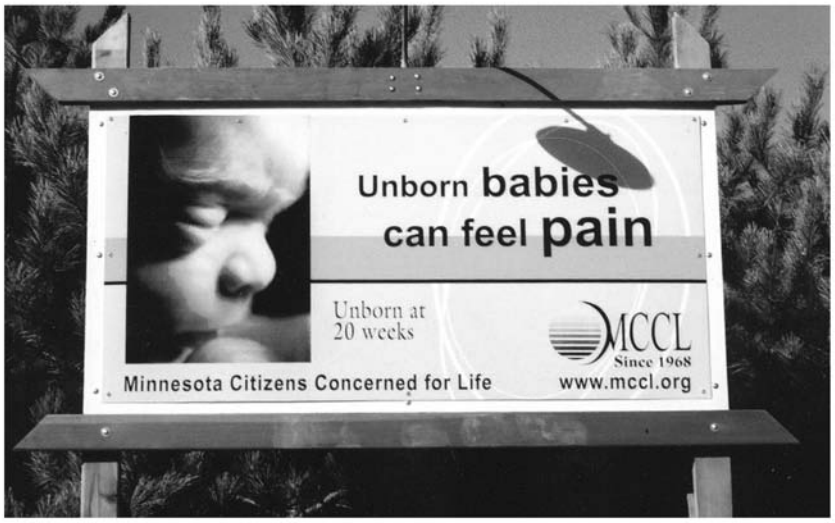
abortions, with pregnancy detection and decision making often occurring late. The women I see in these situations are pregnant and they can't be or don't want to be. They are resolving dilemmas created by circumstances unique to their private lives, and certainly unknown to their critics who judge from afar. I define a dilemma as a situation in which one has to decide between nondesirable options without the luxury of foregoing the decision.

It is in this context that I understand the abortion care that I provide—in the first or second trimester. While their stories might differ, what all pregnant women have in common is the increasing difficulty in abortion access, especially for later abortions. Ironically, it is the lack of access to abortion care that oftentimes delays abortion to the second trimester. A pregnancy in this timeframe is troublesome to those who are in what a friend calls the "mushy middle"—people who approve of abortion access abstractly, but who become conflicted about its specifics, e.g., termination beyond the first trimester. Eighty-five percent of women in the US live in a county where there is no access to abortion and, if later gestational age is taken into account, that access is even more limited. That reality, along with my patients' compelling individual stories, compels me to provide the abortion care that I do, moved to help women in these crisis moments and to prevent the unnecessary health consequences that occur when safe abortion is not available.

The reality is that some women have pregnancies that they did not plan and have no desire to continue and, therefore, they seek abortion—legal or not, safe or not. I believe that it is their right to do so, in the second trimester or the first, that right being rooted in their moral agency as human beings. Thus, I advocate for reproductive justice (RJ).

The RJ movement, as distinct from "reproductive choice," places reproductive health and rights within a social justice and human rights framework. RJ supports the right of individuals to have the children they want, raise the children they have and plan their families through safe, legal access to abortion and contraception. In order to make these rights a reality, the movement recognizes that RJ will only be achieved when all people have the economic, social and political power to make healthy decisions about their bodies, sexuality and reproduction. To be certain, when reproductive justice is present, abortion is available as a choice, but in the RJ framework all reproductive decisions are valued equally. When RJ is a reality, women are empowered to maintain their dignity.

I endeavor to move our world to a place where women have the space and power to make these tough decisions without judgment, coercion or restriction thrust upon them, and are able to do so in a setting of safety and uniform access to all possible reproductive options. It is in this context that I gladly provide first- and second-trimester abortion access for women in support of their humanity, dignity and health. I challenge my peers to do the same. ▪



Antichoice organizations have used the claim that fetuses can feel pain to back up their attempts to limit access to abortion.

Fetal Pain?

By Stuart W.G. Derbyshire, Ph.D.

NEBRASKA RECENTLY PASSED a law to prevent abortion after 20 weeks gestation on the basis that maturing fetuses experience pain and therefore abortion after 20 weeks is cruel and should be banned. Many commentators have observed that the Nebraskan interest in preventing cruelty as a basis to prevent abortion goes beyond the state's legal interest in protecting viable life as a basis to prevent abortion. The latter interest in viability was a key tenet of *Roe v. Wade*. There are at least two problems to untangle. The first relates to the nature of pain and how to decide whether the fetus can ever be said to feel pain. The second relates to the proper role of scientific investigations and discussions in deciding social policy.

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CAN A FETUS FEEL PAIN?

There are two related but separate ways to address whether the fetus feels pain. The first way is to ask what neural structures are necessary for pain and then to ask when those structures develop. Pain is not possible before the necessary neural structures are in place. The second way is to ask, what the psychological content of pain is and then to ask when that psychology develops; pain is not possible before the necessary psychological content is in place.

Examining the development of neural pathways is an attractive approach because it provides substantive answers to the question that can be identified with physical measurements such as images of the brain. In contrast, psychological measures are less substantive. Psychology

involves questions of subjectivity and meaning that cannot be identified with physical measurements. For this reason, most commentary on fetal pain has focused on measurements of neurobiology. Ultimately, however, both neurobiology and psychology have to be addressed together because it is not possible to decide what neural structures are necessary for pain without some conception of "the pain" for which they are necessary.

THE NEURAL BASIS FOR PAIN AND THE NEURAL DEVELOPMENT OF THE FETUS

Since the late 1980s it has been increasingly possible for neuroscientists to look directly at the working brain. Technologies such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) provide structural and functional images of the human brain. That means neuroscientists can observe how the brain looks and also which brain areas are active when the volunteer performs an action, has a thought or feels a sensation such as pain. A series of studies has demonstrated that volunteers experiencing pain activate a large number of neural structures including the lower, subcortical, areas of the brain and the higher, cortical areas of the brain. Imaging studies inform us that these areas are *involved* in pain but not that they are *necessary* for pain. When the cortical areas of the brain are inactivated because of sleep, general anaesthetic or a coma state, however, pain is generally considered impossible. Although contentious, the combined results of imaging experiments and observations of what happens when activity in cortical areas is suppressed lead most neuroscientists to argue that cortical areas are necessary for pain.

The question of fetal pain can therefore be partially addressed by asking when cortical areas become functional in the fetal brain. Around eight weeks gestational age (GA), as the fetal period begins, the developing fetus is approximately 4 cm (1.5 in) long, has similar features to the later stage fetus and has begun to move. At this stage, touching around the mouth will result in movement away, indicating the presence of some early sensory detection. At eight weeks GA, however, the fetal brain is profoundly immature and there are no identifiable cortical areas. Cells in the skin that can detect tissue damage and are necessary for pain also do not develop until at least 10 weeks GA.

After 10 weeks there is evidence of connections between the cells dedicated to detecting tissue damage and subcortical areas. Between 12 and 18 weeks there is the appearance of a developmental cortical structure called the subplate that receives connections from subcortical areas. Some commentators have suggested that this represents the minimally necessary connections for pain. The subplate, however, is a transient brain structure that serves a necessary maturational role. Neurons connect into the subplate and are then held for several weeks before they connect into the mature cortical areas that develop above the subplate. The subplate dissipates and vanishes as the cortical areas mature. Most neuroscientists believe that a maturational structure, such as the subplate, cannot perform a mature function, such as the delivery of pain sensation.

Between 24-32 weeks we can see the substantial growth of connections into cortical areas. Clear evidence of cortical activity during auditory stimulation has been recorded from around 26 weeks GA. Cortical responses have also been recorded in premature neonates of 25 weeks GA following a noxious heel lance. By around 24-26 weeks GA, therefore, it can be assumed that tissue damage causes a cortical response and that the minimal necessary connections for pain are in place.

WHAT IS PAIN?

Typically people do not describe their pain with reference to the activity in cortical areas but with reference to the intensity of the pain and how unpleasant it feels. Pain has a psychological content and is a subjective experience. The International Association for the Study of Pain has officially defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.... Pain is always subjective."

The IASP definition indicates that pain does not have primacy over subjectivity, existing before and *in addition* to subjectivity, but is experienced *through* subjectivity. Pain is a part of knowledge and it is impossible to think of pain without taking account of the whole complex of traits by which we are characterized. By this definition pain is not something that will appear as soon as the required cortical areas are active because pain relies upon a higher cognitive functioning and self awareness that require a protracted period of psychological development. The IASP definition, therefore, appears to rule out the possibility of fetal pain at any gestational age.

There is considerable merit in the IASP definition of pain and in the broader idea

The Nebraska law is deeply problematic, however, for a very different reason. The Nebraska law uses science in an attempt to avoid a difficult social, moral and political question.

that pain is a form of knowledge but there is also a reasonable disquiet in denying a rawer, more primitive, form of pain. A useful distinction might be drawn between just being in pain and *knowing that* I am in pain. Both an older infant and the fetus might be said to be in pain but only the older infant can experience *that he or she is in pain* and explicitly share the condition with others as an acknowledged fact of being. When we experience something we know that it is we who are experiencing it. People do not disappear or drown in sensation but remain self-located within it; our intuition of ourselves as particular things with particular location and experience is opened up by, rather than collapsed into, our senses. It is because we remain ourselves within sensation that we can make choices about how to behave. We may choose to be stoic or protest, for example, when we are injured by others.

If the fetus feels pain then what is felt is something raw and immediate. The pain is, and it is, merely because it is; this simple immediacy constitutes the truth of its existence. If the fetus has any experience at all then it will live those experiences without explicit relationship to them. The experiences will not embed in any general understanding or knowledge system (because no such understanding or system yet exists). The fetus will not know what it is experiencing and with no self-intuition to be opened up by sensation, the fetus will collapse and disappear within sensation. The fetus cannot make choices about how to behave and cannot, for example, launch a protest against the surgeon or choose to be stoic.

It is very difficult to conceive of any feeling that is fully divorced from understanding or knowledge because our everyday sensory experience is always embedded in a context. A touch, for example, might be a warning or the prelude to an embrace or it might be an intrusion (and so a little frightening) or welcome (and so a little exciting) and so on. A touch is never *just* a touch; nobody can experience a touch that is pure and detached from the totality of their being and circumstance. Similarly, nobody hears a pure sound, smells a pure smell or sees a pure object. There is a loss when any sensation or feeling is removed from the situation in which it is attached. What gets lost is the conception of sensation as a subjective experience along with more subtle and complex notions of how social factors and psychological development impinge on the experience. Subjectivity and knowledge contaminate everything that is felt. The fetus may feel something raw and immediate but older infants and adults feel something much more. And once the immediacy of sensation is lost there can be no recovery of innocence.

FETAL PAIN AND ABORTION

The necessary neural structures for pain are developed and functional from about 24-26 weeks GA. Although neural development is continuous and not absolute, based on this evidence fetal pain is not possible before 24 weeks GA. According to the IASP definition, pain requires subjectivity and higher cognitive functions that are not available to the fetus and so pain is not possible at any stage of gestation. Defining pain as something more immediate and raw might have some merit but that makes any fetal pain experience far removed from what is experienced in the older infant and adult.

Based on what is known regarding neural development and pain, the Nebraska law can be viewed as at least a reach both because the timing is off (banning abortion from 20 weeks GA) and because it is unreasonable to equate pain as we typically know it with what the fetus might experience. The Nebraska law is deeply problematic, however, for a very different reason. The Nebraska law uses science in an attempt to avoid a difficult social, moral and political question.

Traditionally the question of abortion has been addressed through arguments about bodily sovereignty and individual rights. At every stage of gestation the fetus is intimately bound up in the woman's physiology and is very much a part of her body. Proponents of abortion argue that nobody should be allowed to force a woman to do something with her body that she does not want to do. On the other hand, opponents of abortion point to the fact that the fetus has the potential to go on and become an independent entity in its own right and nobody should be allowed to prevent that progress. Whether or not the fetus feels pain does not resolve these arguments. If the fetus feels pain then we may still support abortion in the interests of defending bodily sovereignty. Similarly, if the fetus does not feel pain we may still prevent abortion in the interests of defending future life.

The same problem also holds with respect to viability. Technological advances mean that the fetus can survive outside the womb at a slightly earlier age than before but that fact does not resolve the question of abortion. At every stage of pregnancy up to full term it is the case that viability is protected by the fetus remaining alive and inside the womb. When dealing with a wanted pregnancy it is precisely the point to facilitate viability by keeping the fetus inside the womb and deploying medical assistance whenever the baby is born. When dealing with an unwanted pregnancy it is precisely the point to stop viability by removing the fetus from the womb and deploying medical assistance to prevent a live birth whatever the stage of pregnancy.

The neural structures for pain are not available before 24 weeks GA and the psychological experience of pain as we experience it is never available to the fetus. People do not experience pure sensation because they have subjectivity, history and context that are only available postnatally. The fetus does not have subjectivity, history or context and so, if it experiences sensation at all, it must experience pure sensation that is alien to us and will be forever lost through development. None of this can help us decide what we should do with regards to unwanted pregnancy. The issue of unwanted pregnancy involves social, moral and political issues that cannot be resolved by science or technological advance. ■